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ANGINA PECTORIS AND CORONARY OCCLUSION.[1]

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IF any excuse were needed for considering *angina pectoris* and coronary occlusion together tonight, an ample one is provided for you on the chart exhibited in Figure I. Note the heading: it is entirely wrong. These people did not die from *angina pectoris*, whether they had it or not, yet our profession told the Registrar-General that they did. No doubt most of them died from coronary occlusion, a diagnosis which hitherto has been so rarely made that the department includes it under *angina pectoris*! More important still, note the figures 0·44 per 10,000 in 1925, 1·02 in 1930—more than twice as many!

This leaves a wide field for discussion, especially the explanation of the increase. Tonight I am going to say nothing about ætiology, but merely try to discuss some of the clinical aspects.

Though it has long been known that sudden death is at times associated with blockage in the coronary system, it is only in recent years that it has become recognized that occlusion of some part of the coronary system is really a quite common condition, that, further, it is by no means invariably fatal, and, finally, that it can be diagnosed with comparative ease in at least the majority of cases. With this knowledge has come the appreciation of the fact that many of the patients who had in the past been observed to die or to escape death narrowly and haltingly, after presenting the symptoms of *angina pectoris* actually suffered from the disease coronary

[1] Read at a meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association on June 18, 1931.

occlusion. It thus has become of paramount importance to be able to differentiate clearly between those who have the symptom complex only and those who have the particular disease in which the most severe and persistent type of *angina pectoris* is apt to be found. The fact, moreover, that many patients who for varying periods are subject to attacks of *angina pectoris*, finally develop coronary occlusion has tended to focus attention upon the relationship between this symptom and disease of the coronary

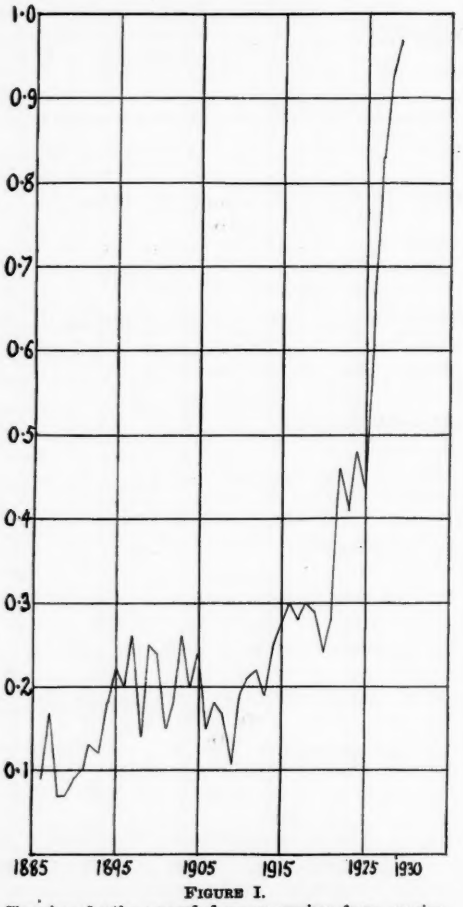

FIGURE I.
Showing deaths recorded as occurring from *angina pectoris* in New South Wales from 1886 to 1929.

system. I intend, therefore, to devote the first part of my communication to a consideration of what we really know about *angina pectoris*, with especial reference to whether we have any criteria as to the gravity of this symptom complex in different cases, and particularly whether we have any means of deciding if a patient subject to *angina pectoris* is in imminent danger of coronary occlusion.

Angina Pectoris.

The term *angina pectoris* was originally coined to describe an attack of cardiac origin in which the outstanding features were agonizing precordial pain, a feeling of severe constriction of the chest and a sense of impending dissolution. Much discussion arose as to the explanation and the fact that disease of the aorta was so often found *post mortem* focused attention on this vessel. Sir Clifford Allbutt was a great protagonist for the aortic origin of *angina pectoris*, regarding the pain as resulting from a sudden rise in endaortic pressure. One hesitates to differ from so great a clinician, but most authorities seem now to be agreed that the chief, if not the only, part played by disease of the aorta is when, owing to structural changes, it is from time to time unable to supply an adequate amount of blood to the coronary vessels and that the most constant pathological factor is a narrowing of the lumen of some part of the coronary system. I think, then, that I am justified in saying that it is now generally conceded that *angina pectoris* is due to sudden and temporary insufficiency in some part of the coronary circulation resulting in deprivation of a corresponding portion of the cardiac muscle of its adequate blood supply. Most of you will, I think, agree that coronary insufficiency may show itself at times by much less dramatic symptoms than those characteristic of the angina of the older physicians.

Such symptoms as comparatively mild precordial pain, a sense of tightness across the chest when walking on an up grade, or again, a sudden sense of complete inability to continue some form of effort will often, in the presence of cardio-vascular disease, provide strong grounds for a diagnosis of coronary disease. There is a tendency to include all such sensations under the caption *angina pectoris*, the great objection to which is that the term is a very terrifying one to the laity on whom it is too often inflicted as a diagnosis which, of course, it is not. For my part I would plead for the exclusion of the expression altogether from the consulting room and the use of some such term as disease of the blood vessels of the heart, painful, spasm of the heart, or something of the kind.

In the absence of definite evidence of organic heart disease we should, of course, be extremely cautious about accepting anything less than the full symptom complex of substernal pain, vice-like gripping of the chest, pains radiating to the neck or arms, especially left, and sense of impending death as indicative of true angina. Vague precordial pains, definite pain and tenderness over the apex and pains radiating to the neck and arms are extremely common in nervous people whose plight will be infinitely worse if we fail to reassure them. When the patient is young we can usually do this unhesitatingly, but when we have any doubt, and especially with a patient in the middle period of life, we may buttress their confidence as well as our own by having recourse to the electrocardiograph. This will detect myocardial disease in so large a percentage of cases that where its findings coincide with the clinical conclusions, we may rest content and remember that pain is far more common in nervous affections of the heart than in organic disease.

While the acceptance of a coronary origin is a great advance in our understanding of *angina pectoris*, there still remains the difficulty of explaining the transitory nature and remarkable intermittency of the attacks. On the basis of purely structural changes, it is difficult to see why effort that will on some occasions induce an attack, will not do so on others, or why exposure to cold after a meal or emotional stress will provoke a seizure without any abnormal physical exertion at all. Moreover, precisely similar changes may be demonstrated *post mortem* in the coronary vessels in the case of one who had had anginal attacks over twenty years, during which steadily advancing structural changes may have been observed in the peripheral vessels, as in the case of another who had died in his first attack.

The special conditions under which the coronary circulation has to function present difficulties in ascribing these variations to vasomotor influences. It is evident that, under normal conditions at any rate, the coronary circulation is in some way shielded from vasomotor stimuli that produce general vascular constriction. Otherwise it would not be possible for them to provide the increased blood supply instantly required to meet the increased output of cardiac energy. It seems probable that the shielding just mentioned is brought about by a depressor mechanism that causes local vasodilatation in times of stress. Reid, in some recent work quoted by East and Bain,[1] is said to have elaborated a theory that there is a depressor mechanism which acts directly on the heart by means of a reflex whose afferent side starts in the aorta. Supported by this hypothesis, Reid appears to uphold Allbutt's theory of the aortic origin of angina.

That, however, spasm of coronary arteries can occur seems to have been settled by some work of Anrep and Segall, also quoted by East and Bain,[1] in which they are said to have demonstrated in the innervated heart of the dog that vagal stimulation causes vasoconstriction of the coronary arteries, while stimulation of the sympathetic causes them to dilate.

Whether spasm of normal coronary vessels can ever be initiated by abnormal efferent nervous stimuli is problematical, but it has long appeared to me extremely probable that under diseased conditions the response to vasomotor stimuli may be altered. That such changes occur in other vessels under pathological conditions is established by the observations that spasmodic fluctuations in the diameter of the retinal blood vessels occur with special frequency in arteriosclerotic subjects. Moreover, the fact that small doses of adrenalin administered to arteriosclerotic subjects produce anginal attacks that nitroglycerine cures, seems to suggest a breakdown in the coronary vasomotor mechanism.

Granting the possibility of spasm, it would seem theoretically possible that the initiating stimuli might originate in the brain, in the upper dorsal region of the cord, where the white rami concerned in the cardiac plexus emerge, or in the viscera, whose viscero-sensory reflexes travel more or less by the same paths as those of the heart. It is interesting, therefore, to recall that the differential diagnosis of *angina pectoris* does in fact mainly rest upon distinguishing pains of cardiac origin from pains derived from one of these three sources, that is, psychic, spinal and visceral. As I am tonight only discussing true angina, I will deal mainly with cases exhibiting frank cardio-vescular disease and try to put before you some clinical evidence indicating that extracardiac lesions may aggravate or increase the frequency of the attacks.

Owing to the difficulty of obtaining any tangible data as to the part played by the emotions, nervous temperament *et cetera*, I will not dwell on this aspect, but remind you of the evidence, probably abundantly available from your own notes, that the exhibition of nervous sedatives in such cases does often reduce the frequenecy of attacks in an amazing fashion.

In regard to the spine, I have seen a number of cases of cardio-vascular disease in which the history suggested that stimuli initiating anginal attacks took their origin from the dorsal nerves. In several of these definite *spondylitis deformans* was demonstrated by X rays. While it is improbable that the actual bony deformities often play any active part in producing pain, it is to be expected that they would from time to time be associated with periarticular inflammation likely to involve the nerves in or about the intervertebral foramina.

In many cases of this type a notable feature in the history is the length of time, often many years, the patients have been subject to the attacks, quite often with long periods of freedom. This seems to suggest that the spinal stimulation induced spasm long before the slowly advancing obliterative changes in the vessels were of themselves of serious moment. It is perhaps worth while reminding you that several cases have recently been recorded in which complete relief of angina has been obtained by injecting the dorsal root ganglia.

The following reports will serve to illustrate the type of case to which I refer:

CASE I: T.S., a male, aged thirty-seven years, for fourteen months had increasingly frequent attacks of pain over his heart and down the lower side of his left arm. Usually they came after slight exertion, such as sweeping the yard, but some have wakened him in the night. Attacks last a few minutes and his wife says he is a bad colour in them. The pulse rate is 72, the arteries show a very slight thickening. The systolic blood pressure is 145 and the diastolic pressure 75 millimetres of mercury. The heart is normal to physical examination and to X ray examination. The electrocardiogram is normal. Hyperæsthesia is present over the fourth and fifth left interspaces from the apex beat to the axilla and over the lower edge of the left *pectoralis* muscle, there is no tenderness on the inner side of the left arm, where he feels pain in attacks. He is markedly tender to pressure anywhere for 5·0 to 7·5 centimetres (two to three inches) to the left of the spine from the second to the fifth dorsal vertebræ. X ray examination shows well marked spondylitis of the cervical vertebræ and less marked changes in the upper dorsal vertebræ.

This case is quoted partly to show how closely *angina pectoris* can be simulated in a case in which the only detectable lesion is a spondylitis, and partly because all the findings were almost identical with those in the case of a man of forty-two years, quoted by Sir James Mackenzie under the heading "Angina Pectoris from Overstrain: Recovery." There was no X ray examination. The man was well and free from pain, except on severe exertion, twenty-two years later.

CASE II: P.R., a male, aged fifty-eight years, had mild albuminuria with occasional hyaline casts, but never any red cells for twenty years. On several occasions in the last fourteen years he had "neuritis" of the left arm, causing pain about the elbow and numbness of the thumb and first and second fingers. Four months ago he had an attack of bronchitis and when coughing on several occasions got a tightness across the upper part of his chest that pulled him up. Lately he has been having attacks like slight angina, a tight feeling across the upper part of the chest (he points to the second and third spaces). He has noticed it several times when out walking, when he has turned sharply to look at anything. He plays golf and has not noticed it then. His heart is a little enlarged, the sounds are good. The systolic blood pressure is 150 and the diastolic pressure 110 millimetres of mercury. X ray examination showed slight hypertrophy of the heart and slight dilatation of the aorta. Very advanced *spondylitis deformans* was present, especially marked in the dorsal region. Twelve months later he died rapidly from some "heart seizure."

This case was of special interest as it is probable that the "neuritis" he had off and on for years was associated with his spondylitis which was unsuspected till a year before his death, as he led an active life and complained of no other pains. The particular movements, coughing and turning his head, that brought on the attacks, suggested that at that stage nerve root irritation was a factor. I lost sight of him during his last year of life.

CASE III: I.F.R., a male, aged fifty-four years, two and a half years ago began to get attacks of pain in the precordia and left axilla, the pain at times going down the left arm. The pain is occasionally severe, more often a dull ache. He may be days without it, but it usually comes after exertion, such as walking smartly and gardening. Sneezing is particularly likely to bring it on. The heart appears slightly enlarged, the second aortic sound definitely accentuated. The pulse rate is 66. The systolic blood pressure is 145 and the diastolic pressure 105 millimetres of mercury. X ray report is that the aorta is rather dilated. The heart is not definitely hypertrophied. Marked spondylitis is present, especially in the dorsal region.

He was given a little bromide and "Luminal" and six weeks later reported that he was much better and had had only one attack when reaching up painting a room. The pulse rate was 70. The systolic blood pressure was 140 and the diastolic pressure 100 millimetres of mercury.

In this case the tendency to have attacks on sneezing and on reaching upwards strongly suggests that the spinal changes were playing a part.

CASE IV: W.J.M., a male, aged sixty-seven years, has pains across the shoulders which seem to make the heart "throb into the left arm" and if he continues what he is doing, he gets a "kind of shortness of breath." The symptoms come on exertion, digging, cleaning his motor car, walking up a hill. He has had them for several years, but they are getting worse. His pulse rate is 76. His arteries are atheromatous. His heart is apparently not enlarged. His systolic blood pressure is 130 and his diastolic pressure 95 millimetres of mercury. Spondylitis and slight osteoarthritis of the whole spine were found on X ray examination.

The symptoms in this case suggest that in the early stages the lesion must have been mainly spinal and that, as arterial disease has developed, the cardiac aspect has become more prominent.

CASE V: Mrs. C., aged forty-two years, for several years past has been subject to some pains in the left side of the neck which have been treated as rheumatism and have from time to time disappeared under treatment. At 9 a.m. she had just got off her bed and stood up when she was seized with a desperate pain over the heart, the left side of the neck and down the left arm. A doctor was brought in from next door and found her of ashen colour, rigid, clinging to the furniture, with her head turned to the left. He gave her morphine at once and she was relieved in about five minutes, but the pain remained in the neck and arm the next day. There was no abnormality to be detected in the heart and the systolic blood pressure was 145 millimetres and the diastolic pressure 90 millimetres of mercury. I saw her two days later and she seemed very well; she had a good colour, her heart sounds were good and her blood pressure the same as above. She was tender to pressure in the left side of the neck posteriorly and about the upper two dorsal spines. X ray examination revealed slight hypertrophy of the heart and slight dilatation of the aorta, also definite osteoarthritic changes in the mid-thoracic region. No gross changes could be seen in the cervical spine. She was given some bromide and "Luminal" for a few weeks, and four months later had had no return of the symptoms.

With such a history it seems difficult to avoid concluding that there was a definite relationship between the spinal condition and the anginal attack.

One of the earliest patients with the visceral type of angina under my care, was a man with advanced aortic disease and anginal attacks. That these always started from the epigastrium, worked upwards and culminated with the belching of wind attracted no special note till he had a severe hæmatemesis followed by a melæna. To my surprise, a few weeks after treatment for a duodenal ulcer his anginal attacks entirely disappeared. I regret that I am unable to complete his history, but three months after the bleeding he was taking exercise consistent with his cardiac disability and was still free from angina.

The following is the only other case of this type that I have recognized.

CASE VI: T.M.R.C., a male, aged sixty-six years, had two brothers who died suddenly from heart disease at sixty-five and sixty-four years of age. He had indigestion for many years. He had some hæmorrhage from a duodenal ulcer three years ago. Two years ago his appendix was removed and the duodenal ulcer was found healed. For eighteen months he has been having typical anginal attacks in which the pain begins below the sternum, works up over the precordium and extends down the left and sometimes into both arms. Attacks come on hurrying, on a hill, after a meal, and particularly wake him about 2 a.m. Belching wind usually terminates an attack. Some attacks have been very severe and he has felt he was dying. Nothing special was revealed on examination, the heart sounds were rather toneless, there was no apparent cardiac enlargement. The systolic blood pressure was 140 and the diastolic pressure 100 millimetres of mercury. The electrocardiogram suggested some degree of myocardial degeneration. It was decided to treat him as a case of duodenal ulcer on a careful diet and alkalis, and for nearly two years since he has been free from attacks and in better health than for years.

In view of the family history and severity of the attacks there is grave reason to fear an ultimate coronary death, but for the time being treatment of the digestion has given striking relief.

The gall bladder is, I think, a frequent offender.

My first case in which a definite relationship was suggested, was that of a man of sixty-nine years, who lived near me and who suffered from typical anginal attacks relieved by nitroglycerine. He had some atheroma, but a fairly satisfactory heart. I was called one night to see him in what was obviously a severe attack of biliary colic. This was relieved by morphine, but jaundice of obstructive type followed. After a few days this cleared up spontaneously, a stone apparently escaping into the duodenum. He promptly lost his angina and, though he is no longer a patient, I frequently see him walking about actively, now about eighty.

In the following cases surgical removal of the gall bladder was carried out.

CASE VII: Mrs. E., aged forty-six years, was seen eight years ago complaining of attacks of pain in the precordial region associated with a cramping feeling and pain going through to the back and down both arms. The duration was three years and the pain was getting worse. It used to come only on exertion, now it sometimes wakes her in the night. Her heart is definitely enlarged. The systolic blood pressure is 200 and the diastolic pressure 115 millimetres of mercury. X ray examination reveals a hypertrophied heart and a dilated aorta. Attacks were somewhat lessened by continuous taking of bromides, but continued till two years later, when she had a severe attack of biliary colic. After the gall bladder was removed the attacks practically ceased, and now, six years later, her daughter tells me that she is leading quite an active life and is no longer troubled with "heart attacks."

CASE VIII: Mrs. D., aged fifty-three years, has had terrific attacks of chest pain with increasing frequency for six months. The pain passes up from the "stomach" to the heart, neck and arms. She cannot move when it comes. Excitement and taking food sometimes start attacks; effort always does so. Sometimes it wakes her. The heart is enlarged, the second sound is accentuated. The pulse rate is 96. The systolic blood pressure is 190 and the diastolic pressure 120 millimetres of mercury. The urine contained much albumin, with hyaline and granular casts. She was very tender over the "gall bladder area." X ray examination revealed hypertrophy of the heart and slight dilatation of the aorta as well as a pathological gall bladder. After removal of a gall bladder filled with calculi she was so free from attacks that she went for a trip to the East. Eighteen months later she died suddenly.

It appears that after removing the accessory spastic factor the organic vascular obliteration proceeded painlessly to occlusion.

CASE IX: J.D., a male, aged fifty-nine years, was for some years under observation for attacks of retrosternal pain which "seemed to grip him about the heart," at the same time he always had a burning pain on the outer side of the middle finger of the left hand. In bad attacks he was unable to move till the attack passed off about three minutes after inhaling amyl nitrite which he carried with him. Quite often the pain began in the finger and spread to the heart. Hurrying or exertion brought on attacks, but they were most likely to come on an hour after dinner, when he always got very distended with wind. X ray examination revealed marked hypertrophy of the heart and very definite dilatation of the aorta. Numerous gall stones were present in the gall bladder.

He gladly accepted the risks of the operation on the chance of relief from his pain. Unfortunately he developed some local peritonitis after removal of the gall bladder and has been left with a tympanitic viscus adherent beneath the scar, apparently the colon. Four months after the operation he reports that he has no attacks in the day time, but quite often wakes at 1.50 a.m. with a feeling of something "creeping up from the scar" and then "away goes the heart" and the acute pain comes in the middle finger. He takes a dose of carminative medicine, belches up a lot of wind and the attack passes off. He has given up amyl nitrite, as the medicine is better.

When we come to prognosis I fear we have to admit that when faced with true *angina pectoris* this is a hazardous proceeding, since, so far as our present knowledge goes, a genuine attack indicates some degree of organic coronary obstruction, and we have no means of measuring its extent or staying its progress. On the other hand, I have endeavoured tonight to combat the idea that, figuratively speaking, we have to fold our hands. We have to remember that Sir James Mackenzie[2] records patients with angina recovering and other cases extending over many years. My own experience has convinced me that under conditions such as I have described, the symptoms are often out of proportion to the coronary disease, and when this is the case occlusion may be postponed for many years, more especially if the accessory lesions are amenable to treatment.

To my mind prognosis is worst when typical angina appears in apparently healthy middle-aged people, more especially when the heart, the arteries and the blood pressure appear to be normal for the age. May not the explanation be that the obliterative process has been allowed to advance right on to the stage of occlusion unhampered by spastic influences?

It must not be forgotten, however, that disease of the coronary vessels, while occasionally the outstanding feature, is after all but a part of a general cardio-vascular disease, so that only a limited number of those with angina actually reach the stage of occlusion. Moreover, there is abundant evidence that gradual obliteration in one part of the coronary circulation can be countered to a large extent by anastomosis with another.

Coronary Occlusion.

Coronary occlusion is the term usually adopted to designate a condition in which a portion of the heart muscle is permanently deprived of its blood supply through blockage of the afferent artery. While embolism does occasionally occur, by far the most common cause is thrombosis of a vessel that has been gradually narrowed by some form of obliterative endarteritis.

As coronary occlusion is a great medical emergency demanding the promptest possible treatment, it is imperative that we should be as skilled as possible in its recognition. Despite all our efforts, in a great many cases death will result either instantaneously or be postponed for a few hours or days. But Levine,[3] who reported 145 cases in 1929, found that about 50% survived the immediate attack. It may be regarded as certain that the future outlook of prospective survivors depends to a very great extent upon the early recognition of their disease and on the treatment carried out. A careful inspection of the necrotic areas in the heart muscle of the various specimens shown tonight should bring home to us the necessity for very complete and prolonged rest as the most essential part of treatment. It should also help to harden our hearts to resist the entreaties of our patients

to allow them up because they feel so well. It is obvious that if any of these patients had survived, it would have been many months before the heart could have rid itself of the mass of inflammatory tissue and have patched up as far as possible the damaged areas. Till then it would surely be desirable that it should expend a minimum amount of energy in carrying on the general circulation.

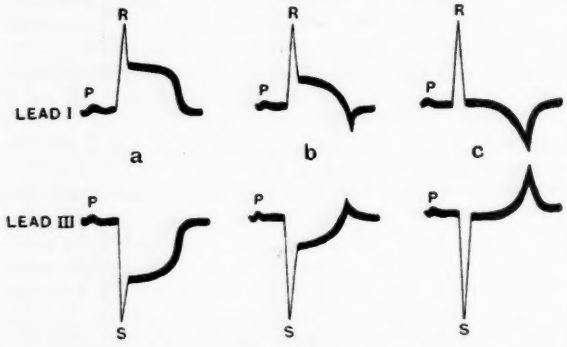

FIGURE II.

A diagram after Parkinson and Bedford, illustrating a form of electrocardiogram characteristic of coronary arterial thrombosis. Immediately after the accident the form is that shown in *a*, but this gives place by transition within a few days or weeks to curves (*b* and *c*) in which T is inverted in lead I or III. Transpose the curves of lead I and lead III in the diagram and they remain equally representative of coronary thrombosis.

In many cases a diagnosis of occlusion is obviously the only possible explanation of the patient's symptoms, but there are occasions when, especially at the onset, quite different lesions are suggested.

When, as most commonly, the onset is with substernal pain, the chief condition requiring exclusion is simple *angina pectoris*. Here the time factor is important, as anginal attacks are usually brief and, in fact, generally over before the arrival of the doctor. I myself suspect that some of the attacks that we still label as rather prolonged and unusually severe anginal seizures, are really minor occlusions and that some of the small patches of fibrosis of the heart muscle often seen *post mortem* originate in this way. In angina the blood pressure tends to rise rather than fall, the attitude is one of rigid fixity, the breathing slow and restrained and the appearance, though pale and anxious, is rarely alarming. In occlusion of the anginoid type, on the other hand, a falling blood pressure is almost invariable, restlessness is characteristic, as also is breathlessness, while the ashen grey or clammy cyanotic facies indicates unmistakably the gravity of the crisis.

Failure to respond to vasodilators supplies valuable diagnostic evidence which is commonly available on arrival at the home of one who suffers from angina. The deliberate use of these drugs for diagnostic purposes, however, would rarely be justifiable and in cases with a falling blood pressure their exhibition might well turn the scale against the patient. In all cases of doubt morphine is a much safer remedy. Like angina, occlusion is apt to occur quite apart from effort, often indeed during sleep. Levine[3] remarks that patients with experience of anginal attacks know at once that this is different, but with several of my patients this was not the case, for they had no anxiety until the usual remedies failed to act.

Quite often vomiting immediately precedes the seizure and sometimes there is purging as well, so that acute indigestion is suspected. The vomiting that so frequently occurs some hours later on, is, I suspect, often due to the morphine employed in treatment.

It is by no means rare for the early pain to be referred to the upper part of the abdomen and to be associated with muscular rigidity and tenderness. In such cases, especially when vomiting is present, an abdominal catastrophe associated with profound shock is apt to be suspected and the differential diagnosis may be further complicated by the knowledge that the particular patient has some such chronic lesion as cholelithiasis. Some degree of pulmonary œdema develops in some stage in most cases, often so rapidly as to be already present on the first examination. In my experience the râles are peculiarly erratic in their distribution, frequently involving only, or mainly, one lung; twice I have found them in the beginning confined to the front of the right lung. In such cases a pulmonary lesion might be first suspected. I can vividly recall having to invoke the aid of morphine to enable me to exclude coronary occlusion and to establish a diagnosis of acute diaphragmatic pleurisy.

Sudden complete unconsciousness for varying periods may usher in an attack and, when there is gross arterial disease, a cerebral vascular catastrophe may be suspected. In one of my cases a pontine hæmorrhage occupied a prominent place in the differential diagnosis for a few moments.

While it may occasionally be difficult at first to exclude an abdominal emergency entirely, in the vast majority of cases a careful, systematic examination of the circulatory system will enable a correct diagnosis to be arrived at on the first examination. The outstanding feature is an abrupt change for the worse from the preexisting cardiovascular state of the patient, hence the great advantage of previous personal knowledge of the patient or a skilfully obtained history, particularly as regards blood pressure.

There is a great fall in the blood pressure, and the pulse, which is often, but by no means always, quickened, is invariably feeble. The apex beat ceases to be either visible or palpable and the first sound becomes toneless and almost or completely inaudible. Gallop rhythm appears early in many cases and is in such surroundings very significant. Later on, in patients who survive, a characteristic feature is the remarkable variation in the pulse, both as regards frequency and regularity. Periods of frequency but little above normal may be succeeded for a few minutes or hours by great rapidity, intermittent attacks of auricular fibrillation are very commonly noted, as also are extra systoles.

I have so far dwelt mainly on the initial signs and symptoms. In cases of doubt or when a wrong diagnosis has been made, decision or correction, as the case may be, will be aided by certain significant after developments.

As an inspection of the specimens exhibited might lead us to anticipate, a pericardial friction rub appears in a considerable number of cases and, when present, may be said to clinch the diagnosis. It usually appears from one to four days after the onset and is generally quite limited in extent.

Some elevation of temperature seems invariably to take place, appearing in about twenty-four hours, ranging usually between 37·2° and 38·3° C. (99° and 101° F.) for the first day or two and gradually settling to normal inside a week. I have not seen a case in which the temperature when taken orally was not at times above normal in the first few days, but Levine[3] says it is sometimes elevated only when taken in the rectum.

As a neutrophile leucocytosis is practically always present, a blood count will often help to fortify the diagnosis, though, of course, it will not exclude the inflamed gall bladder sometimes suggested by a slight icterus which develops in some cases after a day or two. An electrocardiogram has proved to be an extremely valuable accessory aid to diagnosis and it has now been well established that very frequently quite characteristic changes can be recognized when tracings from cases of coronary occlusion are compared with those from normal hearts. On the other hand, since in occasional cases, confirmed *post mortem*, the characteristic changes have been absent, we should not allow our clinical judgement to be outweighed by normal electrocardiographic findings.

I have said so much already that I will deal very briefly with prognosis and treatment.

Seventeen of my patients survived the immediate attack and nine died, but as six were first seen by me when convalescent, the mortality rate works out at about 50% in the acute stage.

Prognosis must of necessity be extremely guarded in even the seemingly mildest cases, owing to the tendency of the clotting to extend to neighbouring branches. A real but quite incalculable danger is entailed in the tendency for a clot to form on the mural endocardium of the ventricle (usually the left) opposite the necrosed area in the wall. Seen *post mortem*, such clots are extremely friable, so that it is not surprising that embolism of the brain and other parts occasionally interrupts an otherwise satisfactory progress. Speaking generally, the patients who seem worst at the onset are most likely to die, but this can be but a very general statement. The general efficiency of the heart apart from the attack has an important bearing on prognosis, for hearts already in a state of unstable equilibrium are unlikely to support the extra burden. Rapid appearance of extensive pulmonary œdema is generally ominous. I have come to regard a low pulse pressure as having very sinister significance and I always have grave forebodings when the blood pressure persists in keeping abnormally low.

Treatment can be summed up in the word rest. In the early stages this can rarely be secured without the use of morphine and in restless cases the drug should be administered unstintingly.

If definite heart failure supervenes, digitalis should be exhibited in the ordinary way. So long as the heart seems to be holding its own, so to speak, more harm than good is likely to result from interfering with it by administering digitalis or any other so-called tonics. Every care will, naturally, be taken to see that the functions of digestion and excretion are carried on in as natural and orderly a fashion as possible.

Believing that minor variations in symptomatology will be best appreciated by the study of actual case histories, I will now have projected on the screen brief notes of cases of coronary occlusion that have come under my personal notice. In preparing these attention has been mainly devoted to the initial symptoms and no attempt has been made to deal with the aspect of the actual ætiology of the arterial lesion, other than to note that sugar was present in the urine in five cases, 20%.

The special vulnerability of diabetics to occlusion is also evident from Levine's[3] figures, which show glycosuria to have been present in 23% of his cases. As the age incidence is about the same as for other conditions, it has been suggested that the diabetes might be regarded as merely another result of the general arterial degeneration. In view, however, of some recent unpublished work on the histology of the arteries of juvenile and elderly diabetics, carried out by Dr. Downie at the Baker Institute in Melbourne, suggesting that there is a special type of diabetic obliterative endarteritis, it seems probable that the coronary lesions may be of the same type.

Dr. Stobo will demonstrate the electrocardiograms which will be shown in relation to some of the cases. In case, however, some of those present are unfamiliar with the subject, he will first briefly indicate the basic principles of electrocardiography.

Case Histories.

CASE X: W.W.K., a male, aged sixty-seven years, had had a sudden sense of suffocation five years ago and collapsed. He recovered his health after some weeks. After running a few steps he had sudden precordial pain and breathlessness and collapsed. He was desperately ill for some days, but gradually improved. Two months later he was breathless on the least effort, his pulse rate was 96, the pulse was soft; his systolic blood pressure was 115 and the diastolic pressure 80 millimetres of mercury. The heart sounds were soft. He continued to improve till one morning three months later, after stooping to pick up something he again experienced violent precordial pain and breathlessness and collapsed. He was found to be yellow-grey in colour, breathless and in great pain, which was somewhat eased by morphine; by the evening he had developed a patch of pericardial friction. His systolic blood pressure was 116 millimetres of mercury. His condition remained precarious for eight or nine days and then he gradually improved. Two months later he was better and his systolic blood pressure was 140 millimetres.

For the last eighteen months he has been able to lead a restricted but useful life.

This case is of special interest, as certainly two, and possibly three, attacks have been survived.

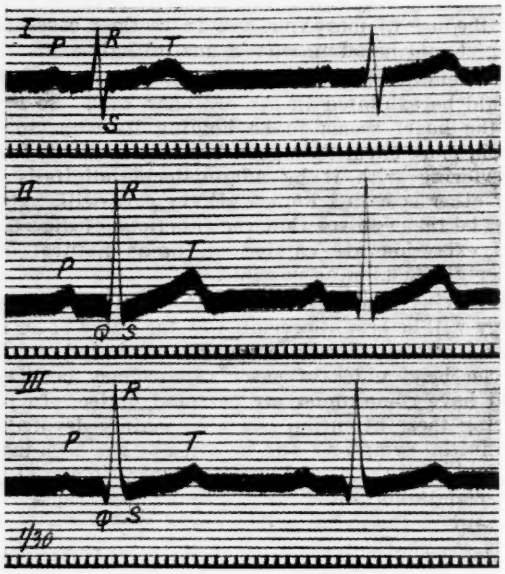

FIGURE III.
Electrocardiograms from the three leads in a normal subject.

CASE XI: P.J.O'C., a male, aged fifty-nine years, caught a cold two weeks ago. Three days ago he felt cold and started to sweat, then he had some pain at the lower end of the sternum. His doctor gave him two injections, but the pain only gradually faded over three days and then shifted to the right side of his chest and is present only on coughing. He was breathless. The heart dulness was obscured and sounds were very distant. The pulse rate was 130. Crepitations were present at both bases. For three days the temperature ranged between 37·9° and 37·5° C. (100·2° and 99·6° F.). The pulse fell to about 100 and breathlessness diminished. Twelve days after the first symptoms occurred, while talking happily, he suddenly collapsed and died. *Post mortem* examination revealed an infarct of the heart wall over the anterior aspect of the left ventricle. A large friable mural clot was attached to the endocardial surface opposite to the infarct.

In this case the history and signs were more suggestive of a pleuritic than a cardiac lesion.

CASE XII: F.H., a male, aged sixty-two years, had no previous ill health, but fainted while doing some outdoor work four months prior to the attack, which first showed itself by a slight but definite attack of angina, the pain passing down both arms. He was kept in bed and next day had another slight transitory attack of angina. His temperature was normal. The following morning he was awakened at 5.30 o'clock with agonizing pain in the chest, going into both arms, especially the left. He was found to be desperately ill, ashen grey colour, dyspnœic and restless. The pulse rate was 140. The heart sounds were very soft. The pain was relieved only after repeated injections of morphine. During the next three days the temperature ranged from 37·6° to 38·6° C. (99·8° to 101·6° F.). He remained restless, clammy and anxious, with feeble heart sounds and rapid pulse. Œdema of the lungs appeared on the second day. On the fourth day vomiting set in and the abdomen became greatly distended. He died on the fifth day.

This was a case with gradual onset, otherwise characteristic. He never looked like rallying. The late abdominal symptoms were interesting, as he seemed to develop peritonitis and possibly had mesenteric thrombosis.

CASE XIII: E.G., a male, aged fifty years, three years ago was seized with "a terribly tired feeling" and fell unconscious. No other illness had occurred, but he was rather breathless on hills in recent years. On stepping off a train one morning he felt a pain in the lower part of the chest which he attributed to indigestion; he called at a chemist's shop and bought some indigestion tablets, but before he had time to take them he pitched forward on to his face unconscious. He quickly recovered and found he had some pain in his chest and that his face was badly cut. He was taken to a metropolitan hospital and his face was stitched up. He sat in a casualty room some hours waiting for a car to be brought for him from a distant suburb, with pain in the chest apparently not bad enough for him to demand relief. He went to bed on arrival home and by next morning the pains had left him. Three days later, while still in bed, the pain came on violently just after dinner, and his left arm felt very tired. His wife says he looked terribly ill and that his pulse disappeared. He was relieved by morphine and stayed in bed for three weeks, after which he saw me at my rooms, looking very well. The pulse rate was 55 and the pulse was regular. The heart was definitely enlarged, the first sound was very soft at the apex and inaudible at the base. The radial arteries were quite good for his age. The systolic blood pressure was 120 and the diastolic pressure 75 millimetres of mercury. The electrocardiogram was typical.

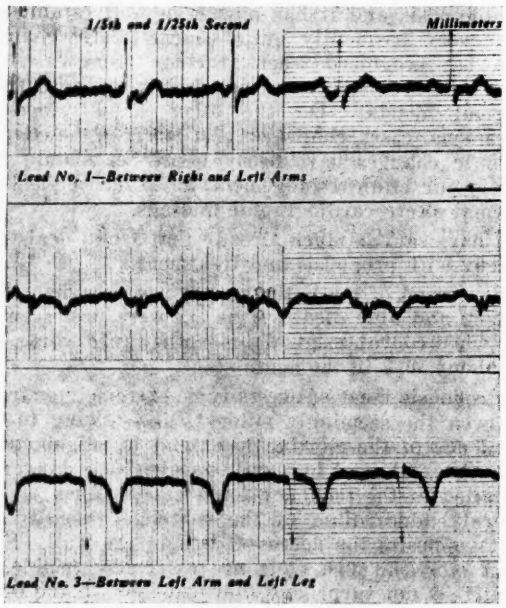

FIGURE IV.
Electrocardiogram in Case XIII.

Special points of interest in this case are the possibility of his having had a slight occlusion three years before, the fact that he became so completely unconscious, and that in spite of a relapse he recovered so satisfactorily. There appears to have been no sense of impending death.

CASE XIV: D.R.S., a male, aged sixty-three years, began to have attacks eighteen months ago, apparently typical of *angina pectoris*—pain and tightness in the precordium

passing into the left arm, lasting a few moments and relieved by nitroglycerine tablets. He was seized just after lunch with pains exactly like his previous ones, but so much more severe that he felt sure he was dying. He was given nitroglycerine, amyl nitrite and 0·015 gramme (one-quarter of a grain) of morphine twice in the four hours before I saw him. He was then ashen grey, obviously in agony, and kept clutching over his heart. He had refused to go to bed and roamed about restlessly, one moment sitting panting on a chair, then walking a few steps and sitting down again. The heart was somewhat enlarged, the first sound was inaudible, the second sound was heard as a faint click, the pulse rate was 136; the pulse was barely perceptible. There were many moist râles. A further 0·03 gramme (half a grain) of morphine gave a little ease and he was got to bed, but died early the next morning.

This was a very typical case with probably very extensive muscular ischæmia.

CASE XV: P.O.R., a male, aged seventy years, had a bad attack of lumbago and sciatica five years ago. For four days he had persistent pains in the left axilla and left pectoral muscle. There was definite tenderness over the left upper intercostal muscles, especially the fourth and fifth. No pleuritic rub could be found. Apart from marked oral sepsis, he apeared quite well otherwise. At 6 a.m. on the fifth day he was seized with violent purging and vomiting, and while in the lavatory had agonizing pain in the chest and felt he was dying. He crawled back to bed on his hands and knees and his doctor found him apparently dying, but he rallied after some morphine. I saw him some hours later, breathless, ashen grey, restless, with heart sounds practically inaudible. The pulse was flickering and its rate was 120. His systolic blood pressure was 100 and the diastolic pressure 85 millimetres of mercury. Moist râles were present at the bases. He vomited violently during my examination and almost died. The pain was somewhat relieved by morphine, but a dull ache remained. He died a few hours later.

It seems possible that this patient had some upper dorsal spondylitis explaining the early pains which were no doubt related to the initial phase of what ended in occlusion. The onset with vomiting and purging is interesting. The low pulse pressure was ominous.

CASE XVI: Mrs. M., aged fifty-seven years, had a sudden sense of weakness in the chest, right arm and right epigastrium twenty months previously. After this she was short of breath for some weeks. Sudden severe pain occurred in the right epigastrium and behind the sternum. This gradually got worse and when seen four days later, she had severe precordial pain radiating into both arms, also severe epigastric pain. The pulse rate was 92 and the systolic blood pressure 100 millimetres of mercury. Extreme tenderness was present over the "gall bladder" area. A few hours later she suddenly stopped talking, became very pale and fell back on the pillow; her son thought she was dead. After she had rallied, the pulse rate was 100, pain and extreme tenderness were present in the epigastric region. Next morning the pulse rate was 116. Respirations numbered 32, crepitations were present throughout both lungs. Epigastric tenderness and slight icterus were present. That night the pulse rate was 100; the pulse was soft. The apex beat was invisible and impalpable. The first sound was very soft. Moist râles were almost confined to the front of the right lung. The colour was moderately good. The temperature was 37·5° C. (99·6° F.). The electrocardiogram a few days later was typical of occlusion. Three months later, after prolonged rest, she is walking about a little.

This was a case which came on so quietly that she did not consult her doctor for four days; when she did, it was hard to decide whether the heart or the gall bladder was at fault, but as she still has occasional epigastric pain and much flatulence, it is quite possible she had both affected.

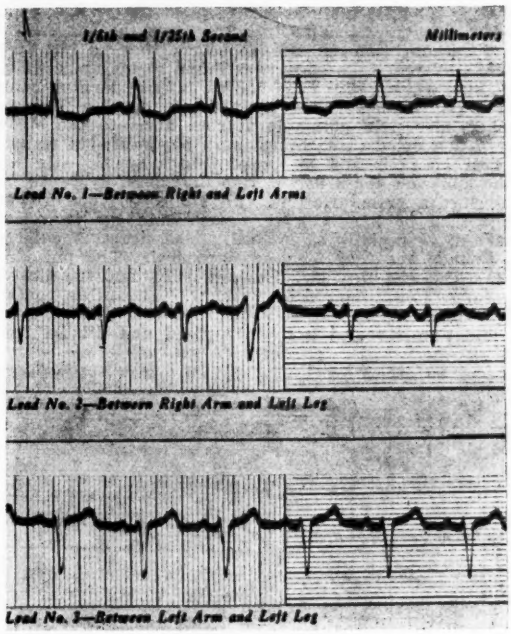
1/5th and 1/25th Second
Millimetres
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE V.
Electrocardiogram in Case XVI.

CASE XVII: J.W., a male, aged forty-seven years, had recently been short of breath on exertion. While in the lavatory he got breathless and could not move, he had to lie down. Later he was removed to hospital in an ambulance and his bowels acted on the way. He was breathless and plethoric. The heart was somewhat enlarged, the sounds were soft and distant, gallop rhythm was present, a systolic murmur was heard in all areas. The pulse rate was 160, the respirations numbered 48, the temperature was 36·4° C. (97° F.). His systolic blood pressure was 105 and the diastolic pressure 70 millimetres of mercury. The temperature was 37·4° C. (99·4° F.) next day and slightly raised for four days. Ten days after the attack there was auricular fibrillation which lasted five days, when regularity was restored.

The notes are rather incomplete in this case, but the patient left hospital able to get about quietly. Pain was apparently not an important feature.

CASE XVIII: Mrs. C., aged sixty years, has had arteriosclerosis and diabetes for many years. Recently her systolic blood pressure was 240 and diastolic pressure 140 millimetres of mercury. During the last four months she has had several attacks of angina lasting a few minutes, the pain often passing into the right arm. One attack lasted an hour. She woke in the night with agonizing precordial pain passing down the right arm; she thought she was dying. She was somewhat relieved by morphine. She vomited a few hours later. Next morning she was pallid, restless, constantly coughing and very breathless. The pulse was thready, its rate was 120. The systolic blood pressure was 110 and the diastolic pressure 90 millimetres of mercury. The first sound was very faint in the mitral area, it was inaudible at the base, numerous moist râles were present all over the front of the chest, especially on the right side; a doubtful pericardial rub was present. She died later in the day.

The fall in blood pressure was remarkable, the pain referred to the right was rather unusual. The

low pulse pressure and early appearance of pulmonary œdema were ominous.

CASE XIX: W.H.M., a male, aged forty-eight years, used to get retrosternal pain going down the left arm two years ago; at times it was so severe that he had to lie down. Six years ago sugar was found in his urine. Eight days ago, when walking, he had an intense burning behind the lower part of the sternum accompanied by great hunger, also pain going down both arms to the elbows. The pain seemed eased by eating a big meal, but returned in half an hour and he broke into a sweat. He went home and sent for a doctor who diagnosed dyspepsia and prescribed a swim. He tried this and nearly got drowned. Since then he has been very weak and the pain keeps returning, especially after food. The heart is enlarged, the sounds are very feeble and muffled. The pulse rate is 80. The systolic blood pressure is 100 and the diastolic pressure 80 millimetres of mercury. The urine contains 1·5% sugar. The electrocardiogram is typical. X ray examination revealed a hypertrophied and dilated heart.

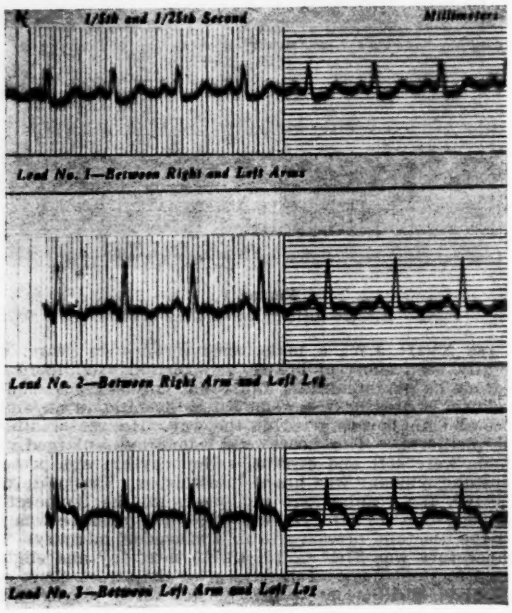
1/5th and 1/25th Second
Millimetres
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE VI.
Electrocardiogram in Case XIX.

He improved after one month in bed, the systolic blood pressure then being 115 and the diastolic 90 millimetres of mercury. He has been carrying on his work cautiously for fifteen months since.

Of special interest is the peculiar feeling of hunger and the fact that he was able to go swimming so soon after, though apparently the effort was nearly too much for him, as he said he barely struggled back after the first few strokes.

CASE XX: E.S., a male, aged forty-nine years, had two attacks of *angina pectoris* close together six years ago. In the second he became blue and pulseless and was expected for some days to die. Ever since then he has felt a tiredness and inability to exert himself in any way. If he persists, he has a sense of discomfort about his heart. He has had *diabetes mellitus* for years. His pulse rate is 80, the pulse is small and regular. The heart sounds are soft, especially the first. The systolic blood pressure is 138 and the diastolic pressure 80 millimetres of mercury. The electrocardiogram indicates myocardial degeneration.

The present state and history seem to indicate clearly that this man had a coronary occlusion six years ago.

CASE XXI: B.K., a female, aged fifty years, was first seen three and a half months after a prolonged attack of angina unrelieved by amyl nitrite. She felt as if both arms would drop off. The systolic blood pressure was 200 and the diastolic pressure 110 millimetres of mercury prior to attack. The heart was found grossly enlarged. The systolic blood pressure was 145 and the diastolic pressure 98 millimetres of mercury. The patient was obese, dyspnœic and cyanotic. Sugar was present in the urine. X ray examination revealed an enlarged heart, a dilated aorta and cervico-dorsal spondylitis. The basal metabolic rate was – 30%. After a further three months in bed the systolic blood pressure was 168 and the diastolic 90 millimetres of mercury. A year later she was much better, the systolic blood pressure being 180 and the diastolic pressure 110 millimetres of mercury. She still had anginal pain on exertion or emotional strain. Four years after the attack the systolic blood pressure was 170 and the diastolic pressure 98 millimetres of mercury. The pain was not so frequent. She has taken thyreoid and "Luminal" continuously.

Despite a gross heart lesion this patient has progressed so well that one suspects that some of the pain may be associated with the spinal lesion.

CASE XXII: P., a male, aged forty-six years, was treated for *angina pectoris* four years ago. He was found to have cardiac hypertrophy. His systolic blood pressure was 200 and the diastolic pressure 120 millimetres of mercury. Three and a half years ago he was in hospital for some time with persistent anginal pain. On discharge his systolic blood pressure was 168 and the diastolic pressure 100 millimetres of mercury, but later the systolic pressure rose to 180 millimetres and the diastolic pressure to 100 millimetres and remained at about that level. He had fleeting pains on walking up an incline. Then he had violent and prolonged pericardial pains after playing bowls; his temperature was raised. His systolic blood pressure was 130 and the diastolic pressure 90 millimetres of mercury. A pericardial rub was present on the fourth day and persisted for a few days. Under treatment and rest the symptoms were relieved, but the systolic and diastolic pressures remained at 150 and 90 millimetres respectively. Three weeks after the onset of an attack he suddenly dropped forward and died in a few minutes.

This man seemed to be making very satisfactory progress, but his blood pressure kept low. He was kept at absolute rest in bed, but, despite this, a further spread evidently took place.

CASE XXIII: T.B.O., a male, aged fifty-two years, gave a history of rather heavy potus. He had a moderate grade of hypertensive arteriosclerosis. The systolic blood pressure was 180 and the diastolic pressure 100 millimetres of mercury. An occasional trace of sugar was found in the urine. One hour after retiring one night, when he had danced till 2 a.m., he woke with severe pain, both thoracic and abdominal, he vomited frequently, he rapidly became seriously ill, pale, breathless and sweating. Auricular fibrillation was found to be present. Under repeated doses of morphine the pain was eased, but did not cease for thirty-six hours. Normal rhythm was then restored. The temperature went up to 38·2° C. (100·8° F.). On the third day the heart was irregular for a few days. The sounds were poor, some congestive signs were present at both bases. On the fourth day the systolic blood pressure was 130 and the diastolic blood pressure 90 millimetres of mercury; gradual recovery occurred after prolonged rest, and the blood pressure remained at about 130 and 90 millimetres of mercury. He was fairly well six months later. The electrocardiogram is typical.

This was a case in which the initial symptoms suggested some form of food poisoning and the fact

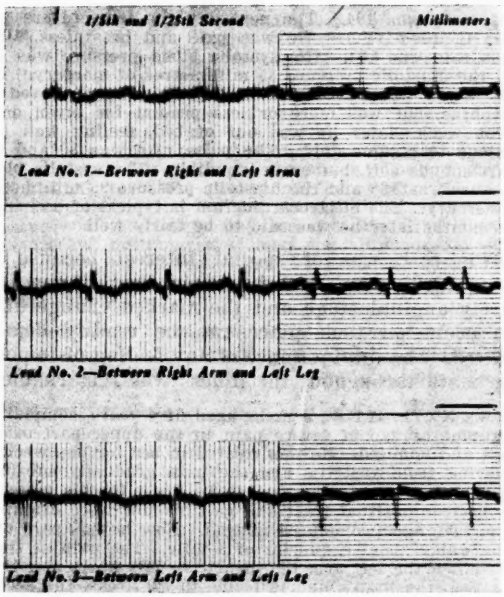
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE VII.
Electrocardiogram in Case XXIII taken on September 2, 1930.

that he had attended a dance and had supper there lent support to this idea. The persistently lowered blood pressure is significant.

CASE XXIV: I.H., a male, aged sixty-six years, had had previous good health. After mowing the lawn late one afternoon he lifted a rather heavy bucket of water and felt some precordial pain extending down his left arm. It persisted and his wife suggested consulting a doctor,

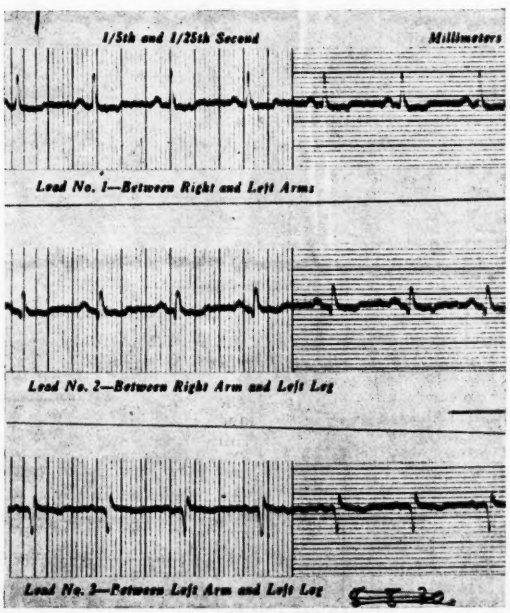
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE VIII.
Electrocardiogram in Case XXIII taken on August 8, 1930.

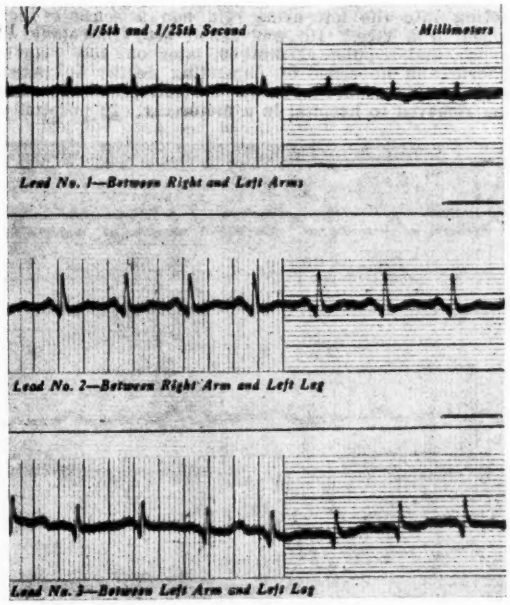
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE IX.
Electrocardiogram in Case XXIII, taken on May 21, 1931.

but he said it was not bad enough. He was kept awake by the pain and about midnight decided to call his doctor, who lived near by. He was found with a dull ache extending from the second left interspace in front to the tip of his left shoulder and down the left arm to the wrists. It was most severe in the front and was not increased by coughing or deep breathing. He was restless and kept changing his position. His temperature was 37·2° C. (99° F.). The pulse rate was 90. The pulse was regular, of good volume and tension. Morphine 0·015 gramme (a quarter of a grain) gave relief, and next morning the temperature was 37·4° C. (99·4° F.), the pulse rate was 100, the blood pressure was 110 and the diastolic pressure 84 millimetres of mercury. Though he was told to keep to his bed, the doctor met him next day returning from the lavatory pale, sweating, complaining of only slight pain, but with a pulse of 150; the pulse was irregular. By the time I saw him next day he had developed a pericardial rub in the sternal region opposite the third rib. He was propped up in bed, was very cheerful, but rather pale. The first sound was very poor; the systolic blood pressure was 110 millimetres and the diastolic pressure 80 millimetres of mercury. There was a good deal of abdominal distension. His progress was moderately satisfactory for six days, the temperature ranging from 37·2° to 38·3° C. (99° to 101° F.) and the pulse from 90 to 100, except on odd occasions, when for half an hour or so it would be rapid and irregular. On the ninth day he died suddenly.

This was a case in which the symptoms developed very slowly. The low blood pressure was rather ominous. It is quite possible that his getting up may have led to a further extension of the process.

CASE XXV: G.I.H., a male, aged sixty-five years, was treated one year and eight months previously for retrosternal pains present intermittently, sometimes waking him at 4 a.m. His arteries were atheromatous. His heart was not enlarged, the sounds were toneless, the rate was 68. The systolic blood pressure was 150 and the diastolic pressure 100 millimetres of mercury. The electrocardiogram showed well marked ventricular preponderance, but was otherwise normal. He collapsed while playing golf and was found lying on a tee with intense precordial pain

shooting into the left arm. He had a sense of being gripped in a vice. He was ashen grey in colour, had dyspnœa and a pulse at first 60, later on, 100. He had euphoria and no sense of impending death; he insisted that he would soon be better. He collapsed twice while being removed to hospital in a motor car. In hospital the pulse rate was 104. The next day the temperature was 37·5° C. (99·6° F.). He was pale and breathless. His pulse rate was 104. His systolic blood pressure was 105 and the diastolic pressure 85 millimetres of mercury. The first sound was almost inaudible. Pain was relieved by morphine, but was more or less present for seven days. Seven weeks later he was out of bed, feeling well and anxious to resume golf. The pulse rate was 80 and the heart sounds soft, but quite inaudible. The systolic blood pressure was 135 and the diastolic pressure 95 millimetres of mercury. The electrocardiogram is typical of occlusion. Six months later he was said to be fairly well.

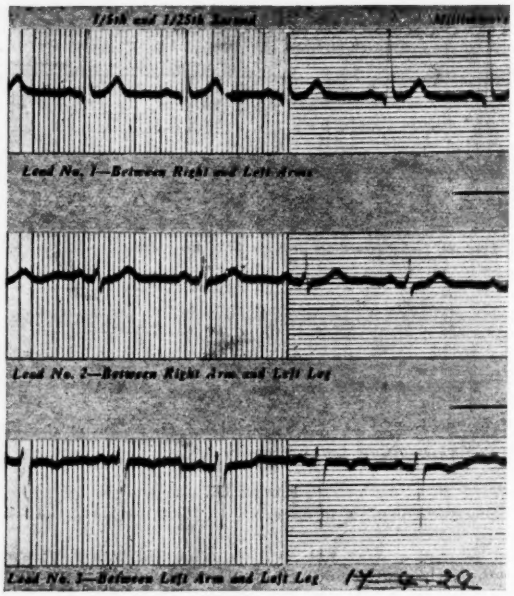

FIGURE X.
Electrocardiogram in Case XXV, taken on April 17, 1929.

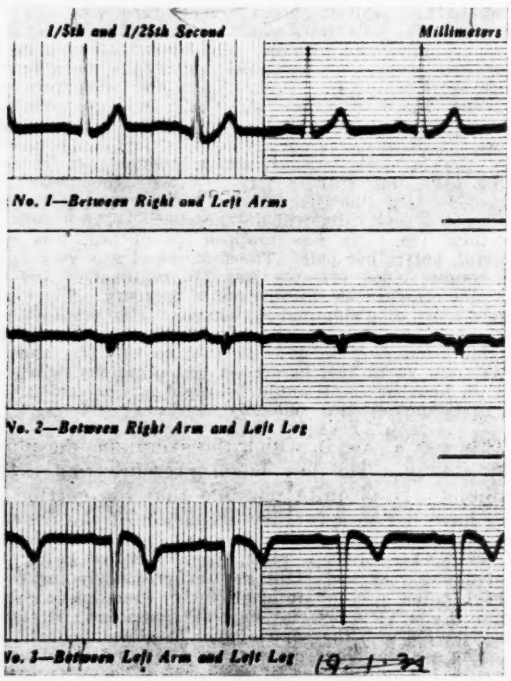
1/5th and 1/25th Second
Millimeters
No. 1—Between Right and Left Arms
No. 2—Between Right Arm and Left Leg
No. 3—Between Left Arm and Left Leg

FIGURE XI.
Electrocardiogram in Case XXV, taken on January 19, 1931.

This case was of special interest because an electrocardiogram had been obtained during the purely anginal stage and the marked change after the occlusion took place can be readily demonstrated. The total absence of any feeling of impending death throughout the illness was remarkable.

CASE XXVI: H.P.G., a male, aged fifty years, complained four months ago of severe pain in the upper part of the chest and vomited. He was treated in bed for one week as suffering from gastritis and the pain gradually subsided. The blood pressure was said to be low. On his recovery the gastro-intestinal tract and gall bladder were examined by X rays and pronounced normal. Now he has pain only if he walks about a quarter of a mile. The pain at times goes down the left arm like neuritis. His heart is a little enlarged, the first sound is feeble in all areas, the second relatively increased. The pulse rate is 60, an occasional extra systole is present. The systolic blood pressure is 130 and the diastolic pressure 105 millimetres of mercury. The electrocardiogram suggests occlusion.

CASE. XXVII: D.O'H, a male, aged seventy-nine years, had a prostatectomy four years before; the systolic blood pressure was then 170 millimetres and the diastolic pressure 100 millimetres of mercury. At 5.30 one morning he suddenly cried "my heart" and fell back unconscious and

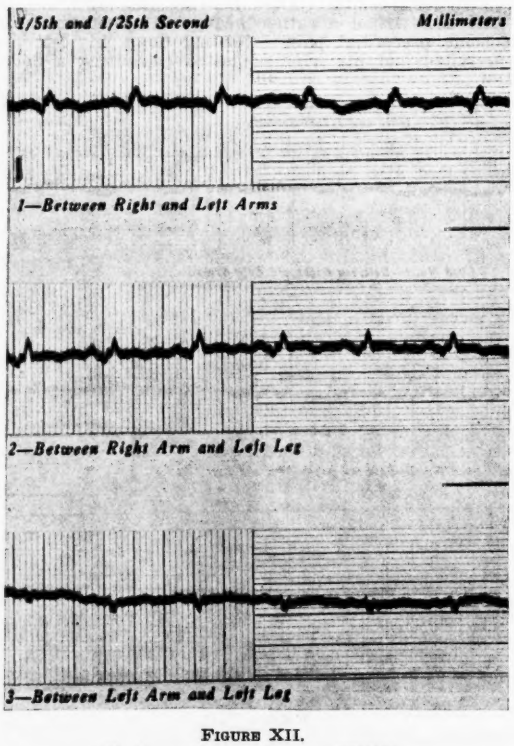
1/5th and 1/25th Second
Millimeters
1—Between Right and Left Arms
2—Between Right Arm and Left Leg
3—Between Left Arm and Left Leg

FIGURE XII.
Electrocardiogram in Case XXVII.

apparently dying. He was cyanosed and clammy, with small, rapid pulse. Shortly afterwards the colour came back and, though he was still unconscious with contracted pupils, the pulse was for a time full and bounding. A little later the pulse failed again and the colour was ashen, but consciousness returned. The pulse was then 104 and soft, the first heart sound was barely audible. From this on he improved. Auricular fibrillation set in and lasted a few hours on the second day. The temperature was 37·8° C. (100° F.) twenty-four hours after the attack and above the normal line for a week. The systolic blood pressure on the fourth day was 135 and the diastolic pressure 100 millimetres of mercury. He made excellent progress for three weeks and then suddenly died. The electrocardiogram on the eighth day was typical of coronary occlusion.

In this case there was no pain at any time; half an hour after the attack began the condition was more suggestive of a pontine than a cardiac lesion.

CASE XXVIII: R., a male, aged sixty-one years, had a "tobacco heart" many years ago; he was cured by abstinence from smoking. He has been a moderately heavy smoker since. He is said to have always had a low blood pressure. Two weeks prior to the attack he had a febrile

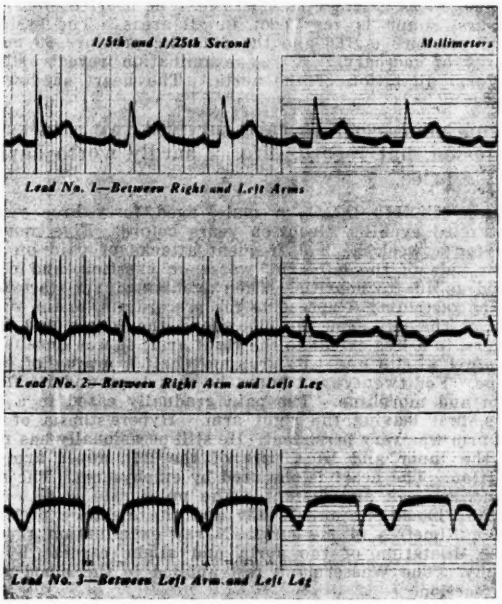
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE XIII.
Electrocardiogram in Case XXVIII.

disturbance regarded as influenza. On the evening preceding the seizure he was not very well; the next morning he had indefinite substernal and precordial discomfort, regarded as indigestion. He carried on his usual work, despite increasing discomfort, till immediately after luncheon, when the pain became excruciating and he had to be taken home. An hour later he was seen lying on the right side with severe pain strictly confined to the precordial area. He was unable to move or take a deep breath. The pulse was soft, its rate was 120. The temperature was 37·2° C. (99° F.). Four hours later the temperature was 38·3° C. (101° F.). The apex beat was invisible and impalpable, the heart sounds were extremely feeble. The systolic blood pressure was 110 and the diastolic pressure 80 millimetres of mercury. Two days later the systolic blood pressure was 114 and the diastolic pressure 80 millimetres of mercury. A pericardial rub was present. The electrocardiogram is characteristic of occlusion. It was two or three weeks before the temperature settled and during this period the condition gave great anxiety, the progress being interrupted by gastro-intestinal disturbance and a sudden, apparently embolic, attack of acute localized pleurisy. Thereafter progress was steady and sure. After complete rest for three months a life of restricted activity was entered on and six months after the onset the condition is extremely satisfactory.

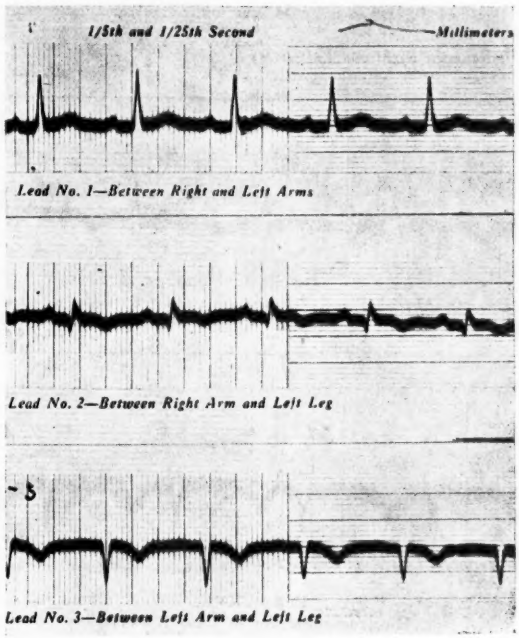
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE XIV.
Electrocardiogram in Case XXVIII.

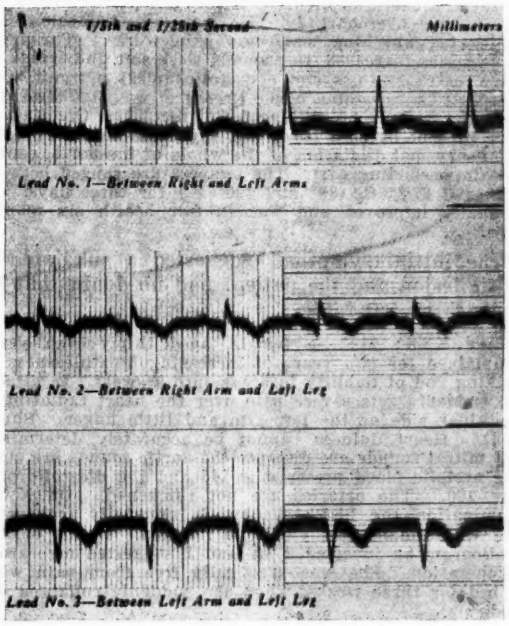
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

FIGURE XV.
Electrocardiogram in Case XXVIII.

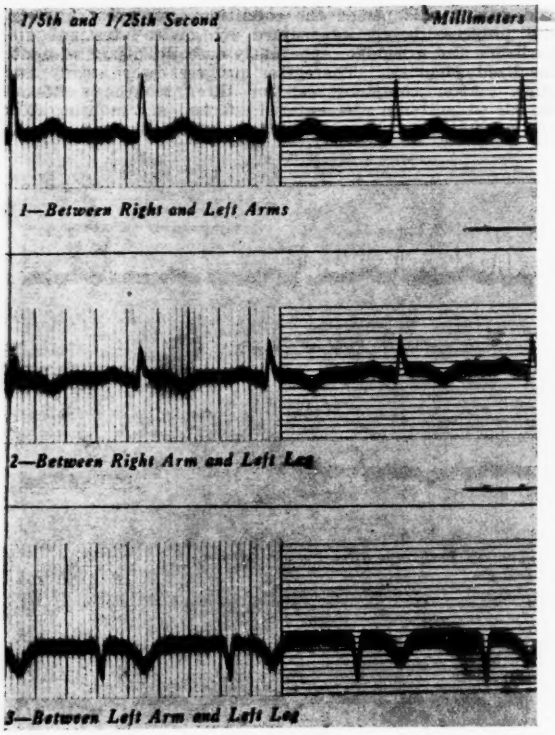
1/5th and 1/25th Second
Millimeters
1—Between Right and Left Arms
2—Between Right Arm and Left Leg
3—Between Left Arm and Left Leg

FIGURE XVI.
Electrocardiogram in Case XXVIII.

CASE XXIX: I.P., a male, aged seventy-one years, was admitted to hospital with a diagnosis of "acute abdominal lesion" which began with gripping pains of continuous character under the breast bone; it felt as if "one of the vessels was overcharged." He was very breathless and swallowing anything made the pain worse. The heart dulness was increased, the sounds were soft and irregular, a few extra systoles and a systolic apical murmur were heard. The systolic blood pressure was 120 and the diastolic pressure 80 millimetres of mercury. Crepitations were present at both bases. X ray examination revealed an aneurysmal dilatation of the whole of the aorta, especially the ascending part. The temperature ranged between 37·2° and 37·8° C. (99° and 100° F.) for three days. He gradually improved and was in fair health six months later.

The initial symptoms suggested a subdiaphragmatic lesion and the patient had no doubt that his stomach was at fault.

CASE XXX: M.F., a female, aged thirty-seven years, complained for two years of increasing breathlessness on walking and of liability to break into a sweat. In attacks she suddenly gets severe pain over the heart radiating to the ulnar side or the left arm and little finger. She is stout. Heart dulness cannot be accurately determined. The mitral sounds are distant; the aortic sounds are clear. The systolic blood pressure is 145 and the diastolic pressure 100. The arteries are not thickened. On X ray examination the heart shadow is unusually transverse. The Wassermann test yields no reaction. In the electrocardiogram an inverted *T* in lead I suggested myocardial degeneration. She was practically free from pain while in bed for three weeks, when she suddenly collapsed and died.

At *post mortem* examination slight hypertrophy of both right and left ventricles was found. The aorta was healthy. Some atheroma of the right coronary artery was found. At the apex of the left ventricle the muscle was six millimetres (a quarter of an inch) thick; the septal wall and much of the left ventricle showed much degenerative change, the muscle appearing necrotic, fibrotic and hæmorrhagic. The left coronary artery showed atheroma and was almost entirely occluded a few centimetres from its origin by a calcareous plaque.

The points of special interest in this case are the apparent absence of any definite attack indicating occlusion, the absence of clinical evidence that the heart was in so precarious a condition. The explanation of such advanced changes in the coronary vessels while the aorta and peripheral vessels were normal is not clear.

CASE XXXI: J.L., a male, aged forty-five years, had chorea at the age of fourteen and rheumatism frequently. Four years ago he had an attack of severe angina unrelieved for two days. Eighteen months later he had another attack of desperate pain which gripped his chest like a vice. The pain did not leave him for some days and his temperature went up to 37·8° C. (100° F.). Since then he frequently has pain over the heart on slight exertion, which goes into the neck and down the left arm. He has to lead a very restricted life. The heart is enlarged, the first sound is very poor in all areas. The systolic blood pressure is 126 and the diastolic pressure 88 millimetres of mercury. X ray examination reveals slight fusiform dilatation of the aorta. The heart shadow is increased.

This man seems to have survived two attacks of occlusion, but regards life as hardly worth having at the price.

CASE XXXII: D.L.T., a male, aged forty-three years, contracted syphilis seventeen years before. Five months ago for a week he had frequent attacks of pain on the inner side of the left arm, worse on exertion, and diagnosed as due to neuritis. Then agonizing pain started in the epigastrium; it spread to the precordium, to the inner side of the left arm, the elbow and the fourth and fifth fingers, to a less extent to the right arm; it was unrelieved by amyl nitrite and 0·03 gramme (half a grain) of morphine. For twenty-four hours he was kept under chloroform and morphine. The pain gradually eased in a few days, first leaving the right arm. Hyperæsthesia of the left arm was very persistent. He still occasionally has pain on the inner and back part of the left upper arm on exertion. The heart is obscured by emphysema. The first sound is weak and distant. The pulse rate is 66. The systolic blood pressure is 130 and the diastolic pressure 105 millimetres of mercury. X ray examination reveals some dilatation of the aorta and slight cardiac hypertrophy. The Wassermann test applied to the blood gave no reaction.

This patient was seen only after he had made a fairly good recovery. The persistence of the pains in the left arm is unusual.

CASE XXXIII: I.G.McL., a male, aged fifty-nine years, while at dinner had a sense of palpitation, shortness of breath and a feeling of constriction in the chest, which soon passed off. A few minutes later it returned and rapidly developed into an apparent anginal attack. His doctor arrived soon afterwards and found him urgently dyspnœic, with a blanched face, complaining of a tearing precordial pain and of his arms being "as heavy as lead," his breathing was shallow and jerky and 44 to the minute; the pulse was rapid and irregular, a few extra systoles were present and the first heart sound was very soft. The systolic blood pressure was 120 and the diastolic pressure 60 millimetres of mercury. The upper part of the abdomen was markedly tympanitic. Amyl nitrite gave no relief, but the pain subsided in about an hour after morphine was administered. Later in the evening the pain returned, but was not so severe. Since then he has gradually improved, but his

systolic blood pressure two months later keeps rather low at about 130 millimetres of mercury and his diastolic pressure at 80 millimetres. He is breathless on exertion and often has slight twinges of pain over his heart on effort or mental excitement. X ray examination reveals a slightly enlarged heart and aorta definitely dilated and elongated upwards. The electrocardiogram shows no abnormality.

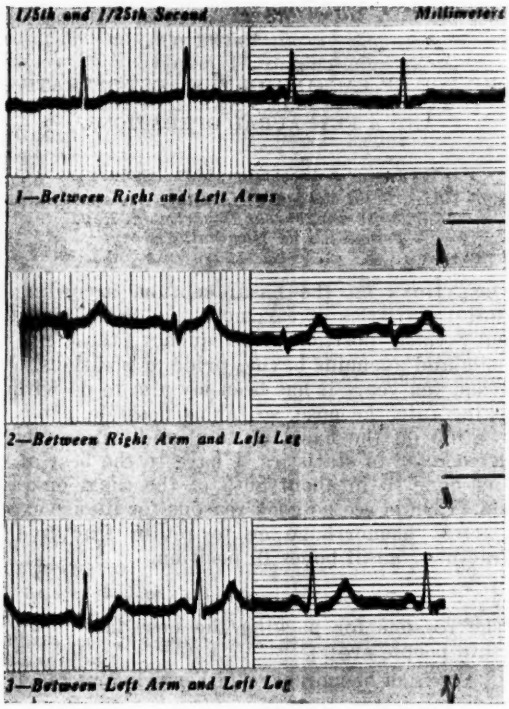
1/5th and 1/25th Second
Millimeters
1—Between Right and Left Arms
2—Between Right Arm and Left Leg
3—Between Left Arm and Left Leg

FIGURE XVII.

Electrocardiogram in Case XXXIII.

Clinically this is a case of occlusion, though the electrocardiogram does not support the diagnosis. It should be mentioned that his blood pressure had been taken on a few occasions prior to the attack and he was told on one occasion that it was 150 millimetres systolic and on another 170 millimetres of mercury.

CASE XXXIV: I.B., a male, aged fifty-six, has for some years been treated for mild diabetes. While walking he was seized with agonizing pains in the lower part of the chest, he felt as if gripped in a vice and about to die; the pain seemed to shoot into the root of his neck. Three days previously he had had a short attack of similar pain, but it rapidly passed off. He was seen soon after his arrival at his home. He was ashy grey, with soft running pulse and in great pain, which was eased by 0·015 gramme (a quarter of a grain) of morphine. Next day the temperature was 38·3° C. (101° F.) and it did not settle to normal for a week. Three days later the heart was definitely hypertrophied, the first sound very soft, and there was a small area of pericardial friction about the size of a five-shilling piece near the apex. The leucocyte count was 16,500, the systolic blood pressure was 120 and the diastolic pressure 85 millimetres of mercury. Fasting blood sugar was 0·29%. The electrocardiogram was typical of coronary occlusion. There was some trouble with abdominal distension for a few days, but he then gradually settled down and now, eight months later, he is very well, but leading a restricted life.

It is interesting to note that the attack of pain three days before the actual seizure, though severe, did not lead this patient to seek medical advice. The initial stage was very alarming, but the subsequent course gave little real anxiety.

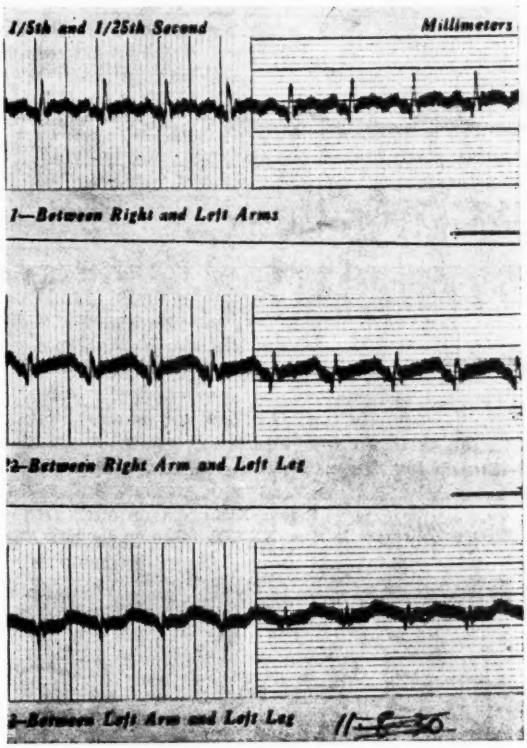
1/5th and 1/25th Second
Millimeters
1—Between Right and Left Arms
2—Between Right Arm and Left Leg
3—Between Left Arm and Left Leg

FIGURE XVIII.

Electrocardiogram in Case XXXIV, taken on August 11, 1930.

CASE XXXV: Mrs. V.H., a female, aged fifty-three years, had an illness which began three months ago with high temperature and collapse; she was in bed a fortnight. Since then she gets frequent attacks in which she feels as if "everything stops in the body"; then something goes up to the head, and she gets a feeling of heavy weight on the top of the head. These seizures are very brief and her own doctor says the heart seems to stop in the attack. There is no palpitation in the attacks now, but at first it used to be terrible. She is stout, and the heart dulness cannot be defined, the sounds are feeble and distant, they number 84 to the minute and are regular. The systolic blood pressure is 115 and the diastolic pressure 85 millimetres of mercury. There were moist râles at the left base. The electrocardiogram suggests myocardial degeneration and coronary occlusion.

I saw the patient three months after her attack. Her account of her illness suggested a febrile attack like influenza. She was very nervous, and owing to her adiposity I was uncertain as to whether she had myocardial disease, but the blood pressure was suspicious. The electrocardiogram was of great assistance.

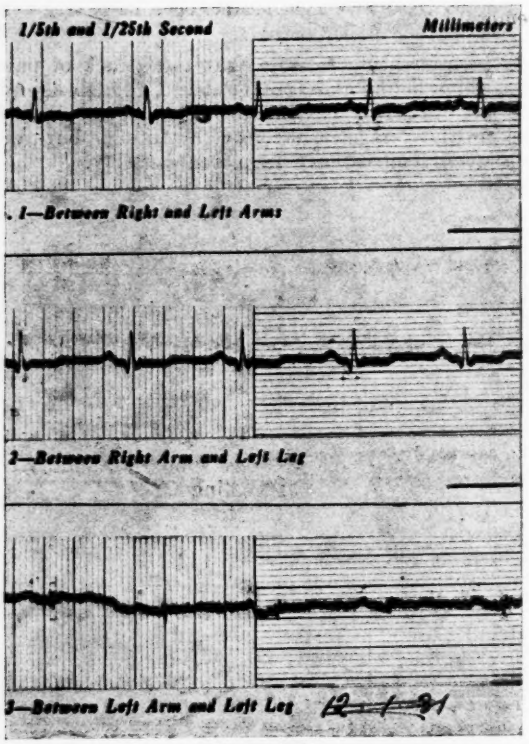
1/5th and 1/25th Second
Millimeters
1—Between Right and Left Arms
2—Between Right Arm and Left Leg
3—Between Left Arm and Left Leg

FIGURE XIX.
Electrocardiogram in Case XXXIV, taken on January 12, 1931.

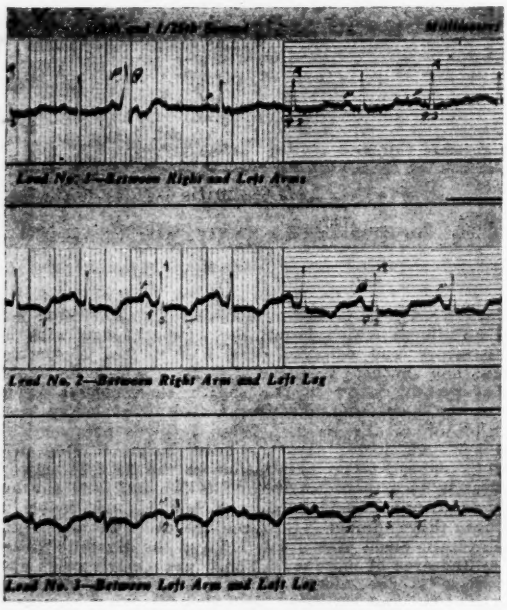

FIGURE XX.
Electrocardiogram in Case XXXV.

Acknowledgements.

In conclusion I wish to express my warmest thanks to numerous colleagues who have collaborated with me in this paper by supplying details about many of the patients I have quoted, the majority of whom I saw only once.

References.

(1) East and Bain: "Recent Advances in Cardiology."
(2) James Mackenzie: "Diseases of the Heart."
(3) Samuel A. Levine: "Coronary Thrombosis," *Medicine*, Volume VIII, Number 3.

THE USE OF PACKING IN THE POST-OPERATIVE TREATMENT OF SOME NASAL OPERATIONS AND A FEW AURAL OPERATIONS.[1]

By JAMES J. WOODBURN, M.B. (Sydney),
Senior Aurist, Saint Vincent's Hospital; Assistant Surgeon, Ear, Nose and Throat Department, Royal Prince Alfred Hospital, Sydney.

OPERATIONS on the nasal septum, according to hospital statistics of ear, nose and throat surgery, occupy third position in frequency, those on the tonsils and adenoids having pride of positions. For the last ten years it has been my aim to do without packing into the nose, so far as possible, after the operation on the nasal septum, and without being able to refer to statistics, I have to the best of my belief done so in about 80% of the cases operated upon. I could see no good reasons for its use except to arrest hæmorrhage or to retain fractured or refractured nasal bones in position.

Reasons for Packing the Nose.

The reasons for packing the nose laid down by certain people are:

1. Arrest of hæmorrhage, either primary, reactionary or secondary.

2. To assist in straightening the nose after a septal operation had been done for considerable deviation of the tip of the nose.

3. To readjust the size of the two nasal cavities and get equality of both—a perfect result.

4. To keep the nasal bones in position when fractured, or when refractured in conjunction with an operation on the deviated septum.

5. When bleeding has been severe at the time of operation for combined operations on the nose, such as an operation for the cure of nasal polypi and a radical cure for ethmoiditis and maxillary sinusitis and the submuco-periosteal resection of the septum at the same time. Packing the nose after such operations will prevent loss of too much blood and subsequent shock, and is in consequence indicated.

6. To prevent a hæmatoma from forming in the septal cavity; but this may happen even if both nares should be tightly packed after operation. A hæmatoma seldom occurs in a septal cavity when the muco-periosteum has been torn or incised in one or more places other than as part of the original incision. Some surgeons make a counter-incision in

[1] Read at a meeting of the Section of Oto-Rhino-Laryngology of the New South Wales Branch of the British Medical Association on May 5, 1931.

the muco-periosteum 2·5 centimetres (an inch) behind the original incision and on the same side.

6. Also in cases of secondary hæmorrhage when it occurs, as it usually does, five to seven days after operation, then it is necessary to pack the nose in the normal way.

Anatomical Considerations.

I will give a brief description of the cells composing the mucous membrane of the nose in order that we may appreciate the damage that can be done to this highly specialized membrane, and show a few photographs of sections of necrosis and regeneration of the epithelium when it is damaged by an attack of "the common cold" in the nose.

The nasal cavities and the sinuses are lined by epithelium of the columnar type. The mucous membrane of the nose is divided into two portions: the olfactory and the respiratory. The olfactory is limited to the area occupied by the superior and middle turbinate bones. This area is lined by two principal types of cells, the columnar non-ciliated and the olfactory cells with large oval nuclei; and also numerous serous glands of the simple tubular type, known as the glands of Bowman, are to be found; and also glomeruli cells of the olfactory nerve endings. Below is a definite and distinct basal membrane. The respiratory area embraces the remaining portion of the nasal cavity, and is lined by columnar ciliated epithelium. Portions of this membrane are thicker and more vascular than the olfactory area, notably over the inferior turbinate bones. Below the surface epithelium are the stellate cells from which the columnar ciliated cells are regenerated and numerous pear-shaped cells exist. These pear-shaped cells regenerate squamous epithelium when the columnar ciliated and stellate cells are damaged or necrosed over an area sufficiently large to prevent normal regeneration. Mucous glands of various shapes are present. A basement membrane lies below all. A deep ulcer of the mucosa of the septum is always healed over by squamous epithelium with fibrous tissue below—it is just scarred tissue covered by squamous epithelium. The principal vessels of the nasal mucosa are as follows:

1. On the lateral wall of the nose: (*a*) *ethmoidalis anterior,* (*b*) *ethmoidalis posterior,* (*c*) *nasalis posteriores laterales.*

2. On the septum: (*a*) *ethmoidalis anterior,* (*b*) *ethmoidalis posterior,* (*c*) *nasalis posteriores septi,* (*d*) *palatina major.*

The microscopical sections shown in the latest year book on diseases of the ear, nose and throat, show the effect of an attack of the common cold on the superficial layers of the nasal mucosa, and an illustration is given in the second picture of regeneration of the highly specialized columnar epithelium from the layer of stellate cells.[1] If such can be the damage by an ordinary cold, how much more severe must be the result of tightly packing the nose for forty-eight hours.

However, according to Gorham and Backer in a series of experiments carried out on the mucous membrane of the antrum after the Caldwell operation had been performed, they are satisfied that complete regeneration of the membrane takes place after five months. Firstly, the red cells or granulation tissue that line the antrum, appear; then a loose type of epithelium grows over the organized tissue and with it the glands reform. This epithelium finally but gradually changes to a perfect pseudo-stratified ciliated columnar type and complete regeneration of epithelium and glands.

[1] Dr. Woodburn showed these illustrations to his audience.

Contraindications for Packing the Nose.

The contraindications for packing the nose are three.

1. When the operation on the septum has been completed and all bleeding has ceased thirty minutes after the gauze containing the cocaine and adrenalin solution has been removed, and again in combined operations on the nose, if there is no bleeding thirty minutes after the original packing has been removed, then there will be no need to pack with bismuth gauze. In a few cases reactionary hæmorrhage will appear later than thirty minutes and if the reactionary hæmorrhage does not cease and is severe, the operator will be called in to pack the nose. Reactionary hæmorrhage occurs mostly from the smaller vessels, for cocaine and adrenalin will not of themselves stop a large vessel from bleeding, so, if reactionary hæmorrhage should occur, it will be mostly from small vessels, and this will give the operator ample time to decide if it is necessary to pack the nose. Ice packs and a dose of morphine if the bleeding is not severe, may suffice to stop a small amount of bleeding. I have set up more bleeding twenty-four hours after an operation when I have removed the bismuth gauze than occurred during the operation the day before. It is such cases as these that make you desist from packing the nose when there is little bleeding at the time of the operation and immediately afterwards.

2. When early atrophic rhinitis is confined to one or both sides, scales may appear after operation, and the patient complains that he did not have scales before the operation. Often with a marked deviation of the septum to one side with a large concavity on the other side, atrophic rhinitis has commenced in the side of the concavity and it is to cure this side of the nose of atrophic rhinitis by diminishing its size as well as to correct the deformity that the operation has been undertaken. These are the cases in which packing should be avoided, if possible, as scales are almost certain to appear in the atrophic side, if the nose be packed for twenty-four or forty-eight hours, and relief of the condition may in this way be impaired. The appearance of scales is due to regeneration of epithelium (squamous or otherwise) before the glands have regenerated and the supply of lubrication (mucus) of a normal consistency has reappeared.

3. When a deflected septum has been operated on to relieve sinus trouble such as frequent attacks of acute antritis with acute frontal sinusitis and acute ethmoiditis on the same side as that on which

the deviation of the septum occurs, gauze packing is contraindicated. Should severe hæmorrhage occur in cases of this type, the nose should be packed, but not for more than eight hours. This will give ample time for the larger vessels to form a clot and the gauze will not be in the nose sufficiently long to aggravate the sinus condition.

Materials Used for Packing.

The commonest forms of packing in use are as follows:

1. Bismuth gauze smeared with vaseline or gauze smeared with an ointment made of bismuth and vaseline.
2. Gauze inserted into the cut off fingers of rubber gloves.
3. Oiled silk rolled into a suitable size and placed in each nostril with the aid of Killian's long speculum.
4. Tampons, such as Dr. Simpson's, which expand when the moisture from the nose acts on them.

Symptoms and Signs Caused by Packing.

The symptoms and signs caused by packing the nose for twenty-four to forty-eight hours are the following: (i) Headache, (ii) restlessness, (iii) slight pyrexia, (iv) enlargement of the nose and swelling in the nasal cavities, (v) marked serous exudation and pus formation, (vi) a number of minute hæmorrhagic areas due to pressure are to be observed when the gauze packing is removed. These areas are due to loss of epithelium and are caused by engorgement of the muco-periosteum and pressure by the gauze which becomes hardened by the nasal secretion and clot, and in many places becomes fixed to the mucous membrane.

Complications Following Packing.

The complications of packing the nose comprise: (i) Loss of smell, (ii) formation of scales and aggravation of atrophic rhinitis, (iii) various forms of sinusitis, (iv) naso-pharyngitis, (v) tonsillitis, (vi) middle ear suppuration and, in rare cases, mastoiditis.

The same remarks apply to operations on the sinuses; that is, if the hæmorrhage ceases shortly after the operation is completed, packing will not be required; an exception occurs after the external frontal operation. In the intranasal or radical operation for cure of maxillary sinusitis, gauze packing will not be required if hæmorrhage ceases after the operation is completed and only if the bleeding persists. After removal of the polypi and exenteration of ethmoidal cells the patient can be treated in the same manner. After removal of spurs on the septum, after removal of the anterior end of the middle turbinate bone or the lower border of the inferior turbinate bone, the patient can be treated without gauze packing.

Use of Gauze in Aural Operations.

I do not believe in packing gauze tightly into the external auditory canal after removal of polypi or of granuloma of the middle ear or of the *membrana tympani* as a routine method.

As most polypi and granulomata are removed while the patient is under ethyl chloride anæsthesia, the operator does not wait long enough for the bleeding to cease, but as a rule packs the ear at once tightly with gauze. This may injure the *membrana tympani* and on rare occasions an ossicle has been found on the end of the gauze after its removal. If the anæsthetic of choice be kelene, the operator should wait to see if hæmorrhage has ceased; if not, he will be able to pack the ear gently with adrenalin gauze and without pain after the patient has "come out" of the anæsthetic. If hæmorrhage ceases, the external ear can be drained with a small gauze wick.

In Heath's operation, the modified radical cure of mastoiditis and middle ear disease, a short drainage tube of suitable thickness can be left in for five days and replaced every second day by tubes smaller in size. Use of gauze is too painful and gauze often requires an anæsthetic for its removal and replacement. When there is no further danger of contraction of the flaps made from the posterior membranous portion of the external canal, then lightly packed gauze can be used for drainage purposes.

Gauze will be found necessary as a packing in the more modern forms of radical operation, but in the old orthodox forms of radical operation gauze for the first ten days can be replaced by a rubber drainage tube to keep the flaps in position. As in Heath's operation, a rubber tube is not so painful to remove and replace as is gauze. In the Schwartze operation, packing tightly by gauze is necessary only at the ends of the incision in order to prevent the skin, fascia and muscles from uniting too readily. If prolific granulations appear on the bone surface, they can be controlled by styptics, silver nitrate or chromic acid, in varied strengths, rather than by tightly packing the whole wound. By packing the mastoid cavity too tightly insufficient drainage is secured, and unhealthy granulations appear.

Reports of Cases.

AMŒBIC DYSENTERY.

By J. BROWN, M.D., D.P.H.,
Honorary Medical Officer, Toowoomba General Hospital.

ON February 3, 1931, Mr. G.P.H., aged thirty-eight years, labourer, of Wallumbilla, Queensland, consulted me for diarrhœa of three months' duration.

Prior to 1915 he was free from diarrhœa. He was till then resident at Wallumbilla, except for eighteen months at Brisbane, where he was born, and for four months in New Zealand in 1914.

From 1915 to 1930 he had occasional attacks of abdominal colic, but no severe diarrhœa. His home was still at Wallumbilla, but occasionally he worked for short periods at various places in the western districts of Queensland and in the vicinity of Clermont, Aramac and Springsure.

In March, 1930, while at Longreach, he had an attack of diarrhœa lasting one week, with abdominal pain and stools which contained blood and mucus. He dieted and took "Painkiller" and "nutmeg and milk" and the attack passed off.

The attack for which he now sought advice began in November, 1930, at Wallumbilla, and the following is his own account, slightly modified, covering a period of three months:

> About 4 a.m. I was awakened by pains all over the abdomen and got up to pass a motion, but while on the way to the lavatory collapsed, and was found two hours later on the ground. I was put to bed and given oil, and about five hours afterwards passed a motion which contained blood and a very little mucus. The pain eased, but fairly bright blood and a little mucus kept coming with all motions. The pain now settled low down on the left side of the abdomen, which was tender to press. The mucus and blood, now darker in colour, increased slightly in quantity, and the tenderness worked up the left side of the abdomen till about two weeks before I consulted you, when a large quantity of greyish material of a honeycomb appearance, which I was told were sloughs from the lining of the bowel, began to come away. This material continued to come away in varying quantity with some blood, and the tenderness went over the abdomen to about half way down the right side. On each of these two occasions (March and November) I had been drinking more or less stagnant water from dams on sheep property during the drought.

He was having remissions lasting not longer than ten days, during which the motions were infrequent and contained no blood. During exacerbations the motions were frequent and contained a large quantity of bright red blood and a little mucus.

He was admitted to the Toowoomba General Hospital looking moderately ill and ill-nourished, and anxious about himself. He had lost weight and his skin was rather dry. There was tenderness on pressure over the large intestine. His temperature, pulse and respirations were normal and continued so throughout. With a diet containing a minimum of residue and colonic lavage with a solution of tannin he improved slightly. He had only one or two motions daily, consisting of a small quantity of greyish liquid, until an exacerbation occurred, when they became more frequent and darker in colour and contained a moderate amount of bright red blood and little mucus. He complained of tenesmus only after lavage.

X ray examination of the lower bowel with an opaque enema disclosed no filling defect in the sigmoid, no evidence of colitis, and only one questionable point, namely, where the descending colon bends forward over the pelvis.

A specimen of fæces was examined by Dr. R. Y. Mathew, of the Commonwealth Health Laboratory, Toowoomba, who reported as follows:

> Blood present, and also blood-stained mucus and many motile organisms of the *Bacillus coli* group. In a wet coverslip preparation many amœbæ were observed. The majority were either sluggishly motile or non-motile, and contained no red blood corpuscles. They were regarded as *Entamœba coli*. Several were larger, were very actively motile, contained red blood corpuscles and were observed to put out pseudopodia and actually to engulf red blood corpuscles. These were definitely identified as the vegetative stage of *Entamœba histolytica*.

Thereupon emetine-bismuth-iodide in keratin coated capsules, 0·06 gramme (one grain), was given three times a day by mouth, and quinine solution was substituted for tannin solution for colonic lavage. No definite improvement in his condition occurred, and the capsules were found unaltered in the stools. On March 4 emetine hydrochloride, 0·03 gramme (half a grain), was therefore given hypodermically twice a day and continued for a week; then emetine-bismuth-iodide, 0·06 gramme, was resumed by mouth. The capsules were not now discovered in the stools. Definite improvement followed closely on the hypodermic administration and was progressive. The stools gradually became brownish and formed, and exacerbations ceased.

On March 10 Dr. Mathew examined a specimen of fæces and reported that no amœbæ were present, but some cysts, probably *Entamœba histolytica*.

On March 26 he examined another specimen and reported that no blood, no mucus, no amœbæ nor cysts were present. This result was confirmed on March 30 after a preliminary dose of magnesium sulphate given by mouth.

The patient was discharged from hospital on April 1, and on June 9 wrote to me as follows:

> I am feeling well and getting stronger, but still have a feeling around the navel as if a sore were being touched. At times this is not present. I am eating well, bowels are regular and motions are normal in colour and consistence.

His weight had also increased.

The chronicity of this infection, which evidently began at Longreach, the remissions, the carrier problem, and the unexpected appearance of amœbic dysentery in this locality render this case of particular interest.

Dr. Mathew's work in connexion with it is very commendable.

Reviews.

A BOOK ON FRACTURES.

"FRACTURES AND THEIR COMPLICATIONS," a book of 415 pages, by Dr. G. E. Wilson, of Toronto,[1] is the work of a practical man of large experience who has devoted much time, considerable thought and a good mechanical sense to the improvement of fracture work. In our opinion he has produced one of the best books on fractures, and we say this in spite of the fact that we do not agree with everything he writes. The author does not irritate us by pretending that everything he does is successful. On the title page he puts this little verse:

> Whoever thinks a faultless piece to see
> Thinks what ne'er was, nor is, nor e'er shall be.

We take it that he intends this to stand, not merely for his book, but for his fracture work also. After what we find in some other books, this modesty is a great relief.

Dr. Wilson is a strong advocate for the fluorescent screen in the reduction of fractures. There is room for two opinions as to the general advisability of this, for it involves a risk to the surgeon, and sometimes to the patient; moreover, to be successful, it must be provided in two planes, horizontal and vertical. The technique of reduction is given as carried out with the viewing screen, but Dr. Wilson recognizes that radioscopy is impracticable to most men, and so he gives always a second set of instructions for technique as carried out without the screen.

He is strongly opposed to forcible passive movement. He thinks massage is useful for the reduction of swelling, but is opposed to exposing fractures involving joints for massage. On the other hand, he believes strongly in the value of active movements carried out by the patient, and in occupational therapy. He lays stress on the great importance of the will to get well.

In regard to splints, he emphasizes the importance of plaster of Paris. He employs the Thomas splint in fractures of the femur, but not according to Thomas. He uses it according to the Hodgen method, making the padded ring superfluous, except as a spreader.

In the treatment of compound fractures he divides the cases, as is proper, into "compound from within" and "compound from without." On the latter he operates to remove destroyed and dangerous tissue and then sutures the tissues loosely. In our opinion this is bad advice. It succeeds in some cases and fails in others. The cases for surgeons to look at and to be perturbed about are the failures. Successes blind them to the failures. The best technique

[1] "Fractures and their Complications," by G. E. Wilson, M.B., F.R.C.S., F.A.C.S.; 1931. London: Baillière, Tindall and Cox. Royal 8vo., pp. 423, with illustrations. Price: 35s. net.

for open fractures is not the one that gives some successes, or many successes and some disasters. The technique that should be aimed at and adopted is a technique that carries no disasters and that saves the most limbs. As far as his book is concerned, Dr. Wilson would seem never to have heard of the Carrel-Dakin method. This is the technique that, properly applied, prevents failure and carries no disasters.

Our author strongly condemns the plating of fractured femurs, which he characterizes as "a very questionable procedure."

The following may be quoted:

> The mistake is frequently made of placing undue importance on the radioscopic appearance of a fracture of the shaft of the femur, for, provided the break is not complicated by joint involvement, if the length is adequately restored it matters little whether they are used to check up the setting or not. . . . Whether the fracture is single or multiple, if the weight-bearing line in the main is seen to pass through the chief pieces, no fear need be felt about the result if the length and position as to rotation are correct, even though a large fragment from one side of the shaft is apparently lying free at a considerable distance from the general line of weight-bearing.

In reference to leg fractures the following may be quoted:

> Nothing has been said about operation in recent simple fractures of both bones of the leg. In our opinion there is no indication for ever making an incision in such cases. . . . To treat an oblique fracture of the shaft of both bones of the leg in a box splint with the knee straight and the foot at right angles is, to say the least, very poor surgery, and if the patient obtains a satisfactory result it will have been achieved in spite of the treatment rather than because of it.

The treatment recommended is to flex the knee and to drop the foot.

In the section dealing with fractures involving the ankle we may quote again his views as to certain practices as bad as they are common.

> It will have been observed that we never wait a few days until the swelling goes down before setting the fracture, as we are of the opinion that the most efficient means of causing the swelling to disappear is to restore the broken bones to their accustomed position. . . . Nor is the presence of blisters containing either blood or serum an indication for postponing the setting, as all they require is sterilization and an adequate dressing. We reduce every Pott's fracture as soon as possible after admission.

People who have suffered ankle fractures involving the posterior lip of the tibia have been given heavy damages against their medical attendants by Australian juries, however near the possible best the result of treatment may be. It is therefore interesting to note that in Dr. Wilson's opinion:

> If there is a wedge-shaped piece off the posterior border of the tibia, the foot must be pulled well forward to obtain the best alignment, but it will usually be found that the upward displacement cannot be completely corrected.

In regard to fractures of the upper extremity we note further his observation that in some cases of Colles's fracture it is not possible to avoid deformity, not due to faulty reduction, but to actual shortening of the radius brought about by the impaction. There can be no doubt that he is right. In his opinion, in the forearm the oblique fractures are those which present the greatest difficulty, and many of them end with a permanent limitation of pronation and supination. Indeed he goes on to state that in adults a fracture of both bones of the forearm usually results in some impairment of function.

An interesting opinion is to be found regarding the treatment of compound fracture of the proximal or middle phalanx of the finger in which the flexor tendons have been severed. He thinks the best thing to do is to suture the *profundus* tendon and to cut away the distal part of the *flexor sublimis*. The deliberate sacrifice of this tendon is more likely to give a useful finger than an attempt to save both tendons.

It will be seen by the various extracts given above that Wilson's is a personal book. It is very far from being a flabby and anæmic compilation. Although, as fracture books go, it is relatively small, it has much more in it than others twice its size. There is no padding. We hope to see it widely used.

Notes on Books, Current Journals and New Appliances.

METEOROLOGY.

"KNOW YOUR OWN WEATHER" is a useful and informative little book which aims at the popularization of the study of meteorology in Australia.[1] Mr. Mares treats his subject simply and clearly, so that not only the student, but the general public, may profit by his experience. The interpretation of weather signs must obviously be of universal interest. The author has contrived in this short space to cover all the necessary ground, yet he gives sufficient explanatory detail for the amateur. Instruments are described, underlying principles of physics are explained, the different types of Australian weather and the interpretation of signs from which weather may be forecasted are discussed. Moreover, advice is given to mariners and to aviators. There is an excellent glossary, and the sketches and diagrams are clear and well explained. Perhaps the photographs, especially of typical cloud formations, could be better and more numerous. The book is, however, well worthy of study.

THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY.

THE second number of the first volume of *The Australian and New Zealand Journal of Surgery* (September, 1931) has been published. This journal, replacing *The Journal of the College of Surgeons of Australasia*, is published quarterly. The articles appearing in this number are of considerable interest. Professor Harold R. Dew contributes a long article on *Echinococcus alveolaris*. This article is referred to elsewhere in this issue of THE MEDICAL JOURNAL OF AUSTRALIA. Professor Ernest W. Hey Groves, of the University of Bristol, England, writes on "Nature and Art in the Healing of Fractures." Dr. Meurice Sinclair, well known to surgeons in France during the Great War, discusses the use of the Thomas splint in fracture of the long bones. Dr. Sinclair's article is an "amplification" of an article by Dr. C. E. Corlette, published in *The Journal of the College of Surgeons of Australasia* of March, 1930. An important contribution comes from the pen of Dr. P. N. Walker-Taylor. He deals with transplantation of the ureters into the intestine and gives details of the way in which he evolved what he calls the "aseptic irreversible tunnel technique." It is intended to refer in a subsequent issue to this important piece of work. Dr. Frank L. Apperly writes on "The Spastic Colon as a Cause of Chronic Abdominal Pain," and Dr. Julian Smith contributes an article on cancer of the tongue, in which he makes particular reference to the use of radium. The case reports include an interesting account of an operation on a large cirsoid aneurysm of the scalp by Dr. Henry Searby. Dr. Searby's contribution is illustrated in such a way that the successive steps of the operation are made clear. Dr. Ewen Downie gives an account of adherent pericardium treated by cardiolysis and Dr. Keith Ross reports several cases of carcinoma of the jejunum and ileum.

[1] "Know Your Own Weather: Popular Studies in Australian Meteorology," by D. J. Mares, F.R.M.S.; 1930. Australia: Angus and Robertson. Demy 8vo., pp. 138, with illustrations.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 26, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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ECHINOCOCCUS ALVEOLARIS.

THE story of hydatid disease in its broad outlines is known to most medical practitioners in Australia; from their earliest student days they have formed mental pictures of the life history of *Tænia echinococcus* and in the surgical wards of the teaching hospitals they have seen hydatid cysts in all parts of the human body. The non-medical members of the community know that Australian practitioners have unusual opportunities for studying the disease and expect Australian surgeons to be expert in its treatment. Medical practitioners who served at one of the base hospitals in France will remember the blunt and determined refusal of a "digger" to have his kidney removed for hydatid disease by one of the foremost British surgeons because hydatids were "a curiosity to Englishmen," while their removal was "like shelling peas" to Australian surgeons. But the whole story of hydatid disease has not been told. The lesion known as *Echinococcus alveolaris* takes the form of a more or less firm tumour composed of a tough stroma surrounding innumerable, almost microscopical alveolar cavities containing gelatinous material or small cysts. Up to the present time no instance (with one possible exception) has been reported as occurring outside a narrow zone in Europe—South Germany, the Tyrol, Bavaria and Serbia. In these countries the alveolar form of the disease is almost as common as the form with which Australian medical practitioners are familiar. On account of the peculiar characteristics of the lesion and the limited area of its occurrence in Europe, the view was advanced that there are two distinct parasites—one responsible for the usual form known to Australians, and the other giving rise to the alveolar form. One of the upholders of this dualistic school has been no less an authority than Dévé, of Rouen. As long ago as 1925 Professor Harold R. Dew, whose work on hydatid disease is so well and favourably known, sided with the unicists. He based his deductions partly on analogy and partly on a study of the pleomorphism of hydatid disease as seen in domesticated animals. He believes that nothing has as yet been demonstrated in the pathology of any hydatid lesion that cannot be adequately explained by the occurrence of parasitic variation. He has now been fortunate enough to discover in Australia a true case of *Echinococcus alveolaris* and has published it in full detail in the September issue of *The Australian and New Zealand Journal of Surgery*. Professor Dew points out that, although Wernicke reported a case in South America, it was of doubtful authenticity, because the birthplace of the patient was not known with certainty. Professor Dew's patient was a man of sixty-seven who had spent the whole of his life in Victoria and Tasmania. The lesions found at *post mortem* examination were typical of *Echinococcus alveolaris* and the diagnosis was confirmed by Dévé, to whom specimens were sent. The significance of the findings cannot be over-estimated, for they undoubtedly do away with one of the main supports of the argument of the dualistic school. It must also be conceded that Professor Dew is right when he states that careful search in areas outside the narrow European zone would surely reveal other cases of the alveolar form. At the same time, he points out that the unicists have not been able to give a complete explanation of the peculiar pathological appearance of the lesion. His discussion on the pathological appearances is most interesting and he holds that the lesion in the case described by him exhibits features common to both

the alveolar form and *Echinococcus multilocularis* (multicystic hydatid lesions). This leads him to the conclusion that transitional forms between all the morphological variants of hydatid disease can occur and that there is no need to postulate the existence of two specific tæniæ. He asks the dualists what he describes as a pertinent and at present unanswerable question: If the lesion is a specific one, confined to man, how can the life cycle be completed by passage through a definitive host and the parasite be prevented from becoming extinct?

There is no need to discuss Professor Dew's communication further. Those who are interested in the pathology of hydatid disease (and we maintain that all Australian medical practitioners should be interested), will study his case report and follow his arguments. There can be no doubt that he shows the need for further careful clinical observation and *post mortem* examination on every possible occasion. Perhaps the Hydatid Registry of the Royal Australasian College of Surgeons will be an incentive in this direction. It should be. Emphasis must also be laid on Professor Dew's statement that carefully conceived and fully completed feeding experiments will solve the problem. Australian medical practitioners have abundant opportunities for studying hydatid disease. The non-medical members of the community expect them to be expert in its recognition and treatment. Scientific workers in parts of the world where hydatid disease does not exist, will expect Australians to complete the story of the disease in all its aspects. They can do it, if they will.

Current Comment.

ANGINA PECTORIS.

ELSEWHERE in this issue will be found a discussion on the theories concerning the pathological conditions of which *angina pectoris* might be a symptom. Lately, the "coronary" theory of causation has gained considerable favour at the expense of Allbutt's "aortic" theory. Francis C. Wood and Charles C. Wolferth, assisted by Mary M. Livezey, recently undertook an investigation with the object of testing the validity of the coronary theory.[1] Electrocardiographic studies were made of thirty patients suffering from *angina pectoris*. Records were taken before, during and after the paroxysms. In six cases the paroxysms occurred spontaneously; in the remaining twenty-four they were induced deliberately by means of exertion. Controls consisted of 100 normal subjects; 50 patients suffering from cardio-vascular disease of various kinds; twelve patients who had, at some time, suffered from *angina pectoris*, but who did not become affected as a result of the prescribed exertion; and four patients subject to paroxysms of pain due to some extracardiac cause. Exertion resulted in an alteration of the electrocardiographic appearances in all instances. In the records of the controls, with few exceptions, inverted *T* waves tended to become upright, and upright *T* waves tended to increase in height. There was usually no pronounced alteration in the *Q R S* complex. Electrocardiographic examination of the patients suffering from *angina pectoris*, in five instances, revealed changes similar to those caused by exertion in the control group; in the remaining fifteen instances the alterations in the ventricular complexes occurring during the pain were quite different; the *T* wave tended to become flattened, inverted or diphasic. The severity of the pain did not seem to have any influence on the electrocardiographic appearances. Wood and Wolferth suggest that the changes in the electrocardiogram are due to some intrinsic changes that take place in the heart itself, and are responsible for the paroxysm.

Experiments were undertaken with the object of learning whether occlusion of a coronary artery would result in the development of electrocardiographic changes comparable with those observed in cases of *angina pectoris*, and whether, when the obstruction was removed, the heart could recover so that its associated electrical phenomena would return to their former state. Dogs and cats used in the experiments were anæsthetized by the administration of iso-amyl-ethyl barbituric acid. Occlusion of a large coronary artery rapidly caused a remarkable alteration in cardiac contraction. Cyanosis of the area supplied by the vessel appeared, and, after a minute or two, the ventricle commenced to dilate. If the vascular constriction was not soon relieved, ventricular fibrillation ensued. Providing fibrillation had not commenced, the removal of the constriction was followed by recovery of the heart muscle. There were striking electrocardiographic changes, similar to those revealed by the examination of patients suffering from *angina pectoris*; they were most pronounced in those instances in which the heart had been subjected to previous experimentation and had thus suffered some myocardial damage. In some cases, however, electrocardiographic records taken while the vessels were occluded, were no different from those taken before the commencement of the experiment. In each instance the electrocardiographic appearances observed after the removal of the constriction were the same as those observed before the commencement of the experiment. It is notable that the electrocardiograms of animals were apt to become

[1] *Archives of Internal Medicine*, March, 1931.

abnormal as a result of previous experiments. The protagonists of the "aortic" theory remark that coronary occlusion must always cause arrhythmia, which does not occur in association with *angina pectoris*. But in Wood and Wolferth's experiments arrhythmia occurred only when ventricular fibrillation was imminent, or when the heart muscle was irritated during manipulation. Accompanying *angina pectoris* there is usually a rise in arterial blood pressure; in these experiments there was usually a fall in blood pressure; Wood and Wolferth remark in this regard that the blood pressure of an anæsthetized animal cannot be compared with that of a conscious man. The sceptical reader will, with every justification, suggest that other phenomena are not comparable.

The factors that seemed to be of importance in the production of changes in the electrocardiogram were: the site and size of the occluded vessel (the most pronounced changes followed obstruction of vessels on the posterior wall of the heart), the state of the heart muscle before the commencement of the experiment, the duration of the occlusion, and, possibly, the obstruction of veins accompanying the occluded artery. Wood and Wolferth conclude that the evidence they obtained was compatible with the hypothesis that the majority of attacks of *angina pectoris* are due to coronary obstruction; but that it did not eliminate the possibility that pain may be produced by other factors.

The results of these experiments are interesting; but they do not seem to bring the problem of the ætiology of *angina pectoris* any nearer solution. There can be no *angina pectoris* without pain, and the cause of *angina pectoris* cannot be found by experiment that does not involve the production of pain. The anæsthetized animal feels no pain. Even if it were practicable to occlude a coronary artery in an unanæsthetized animal, it would not be possible to obtain information concerning the exact distribution of the pain thereby produced.

Evidently a rapid degeneration of the myocardium followed operations on the hearts of animals employed by Wood and Wolferth in their experiments. This may have been due to manipulation, post-operative pericardial adhesions, temporary coronary obstruction, or the poisonous action of the anæsthetic. If it was due to coronary obstruction, it might be used as an argument against the coronary theory. The difficulty of correlating the results of experiments on laboratory animals with the phenomena associated with an attack of *angina pectoris* is very great; it might even be said that such correlation is impossible.

A NEW ANTIBACTERIAL AGENT.

SOME experimental results of exceptional interest are contained in two papers recently published from the Rockefeller Institute by O. T. Avery and R. Dubos.[1] They concern the influence of an enzyme obtained from a saprophytic soil bacillus, on the specific carbohydrate of Type III pneumococci and on the course of experimental infections with pneumococci of this type.

Earlier work from the same laboratories has shown that the nature of the specific carbohydrate contained in the capsule of virulent pneumococci determines the serological type of the organism. An antiserum which is precipitated with the specific carbohydrate of Type I, agglutinates pneumococci of that type and protects mice against experimental infection with the strain, but shows none of these activities against pneumococci of other types. Further, it has been shown that the specific carbohydrate, which in itself is entirely non-toxic, possesses an "aggressin" effect in that its addition to an otherwise innocuous mixture of virulent pneumococci and antiserum may render the mixture fatal to the inoculated animal. The specific carbohydrate thus occupies a central position in the immunological reactions which determine the outcome of pneumococcal infections.

The pneumococcal specific polysaccharides have proved very resistant to enzymatic hydrolysis, but the soil bacillus obtained by Avery and Dubos produces an enzyme which has a rapid destructive action against the polysaccharide of Type III pneumococci. The action is probably hydrolytic and is uniquely directed against this one type of bacterial polysaccharide. In some respects the enzyme seems to be even more specific in its action than the type specific antibody. As well as destroying the purified polysaccharide, the enzyme appears to be able to dissolve or inhibit the formation of the capsule in living pneumococci. Type III pneumococci will grow quite well in mixed culture with the soil bacillus, but the cocci developing in such cultures are unencapsuled and are not agglutinated by specific serum.

If a sterile enzyme preparation is added to a lethal dose of pneumococci and the mixture injected intraperitoneally into mice, no effect is evident with Types I and II, but with Type III a high degree of protection is afforded. In one experiment, for instance, mice were protected against one million lethal doses by 0·1 cubic centimetre of an enzyme preparation. The enzyme has curative as well as protective qualities. Mice may be protected by its injection as late as eighteen hours after injection of a lethal dose of living pneumococci.

In addition to illustrating again the dominant part played by the specific carbohydrate in determining the outcome of infection, these results seem to indicate the possibility of developing an entirely new method of therapeutic attack on the bacterial infections. It is particularly interesting that these initial experiments concern Type III pneumococci, against which it is notoriously difficult to obtain an effective antibody response. The use of such hydrolytic enzymes may, of course, remain a mere laboratory curiosity, but at the moment it seems to open a new and most promising field for experimental and eventually clinical research.

[1] *Journal of Experimental Medicine*, July 1, 1931.

Abstracts from Current Medical Literature.

RADIOLOGY.

Chronic Hyperplastic Tuberculosis of the Colon.

SAMUEL J. GOLDFARB AND MARCY L. SUSMAN (*American Journal of Roentgenology and Radium Therapy*, March, 1931) describe chronic hyperplastic tuberculosis of the colon. They first give a classification of tuberculosis of the colon into: (i) ulcerating type, (ii) cicatricial type from healing of annular ulceration of the bowel, (ii) enteroperitoneal type with definite tendency to softening and suppuration, (iv) chronic hyperplastic type. The last is the least common and least known. It often consists of a primary lesion without evidence of the disease elsewhere. It usually occurs in the middle period of life, and is generally localized to one portion of the colon, though there may be two or three distinct lesions, and rarely the so-called "gas-pipe" colon is seen. The authors quote statistics of Mummery. In 48 cases the lesions were in the caecum, in 39 in the caecum and ascending colon, in six in the sigmoid flexure, in four in the whole colon and three in the caecum, ascending and transverse colons. In only 24 of one hundred cases was tuberculosis found elsewhere. There is tumour formation accompanied by stricture of the bowel, and the disease is essentially chronic, with fibrous tissue formation rather than caseation or ulceration. The mucous membrane may be intact. Chronic non-specific infectious granuloma of the colon can closely resemble the condition, both clinically, Röntgenologically and pathologically. The Röntgen appearance of the hyperplastic type of tuberculosis is a filling defect with spastic manifestations. The barium enema shows the caecum and ascending colon irregularly filled and decidedly narrow. The normal haustrations are absent and Bauhin's valve is incompetent. Assman asserts that the Röntgen appearance is not sufficient for a differential diagnosis.

The Discovery of Röntgen Rays.

OTTO GLASSER (*American Journal of Roentgenology and Radium Therapy*, April, 1931) states that thirty-five years is so long ago that it is difficult to obtain authentic facts from original sources bearing directly upon the manner in which the Röntgen rays were first discovered. There are still persons living who had personal and intimate contact with Röntgen and who can relate their experiences with authority. According to statements by these persons, the phenomenon was first manifested by the fluorescence of a small barium platinum cyanide screen under the influence of rays emitted from an excited Crookes's tube. Sylvanus P. Thomson, well known English physicist, lectured at St. Martin's Town Hall, London, on November 5, 1897. This lecture agrees with the report of many others and must have related events as they actually occurred. Thompson's report definitely establishes the fluorescence of the screen as the first observed phenomenon, and gives a denial to many stories and fables, the principal one of which was that Röntgen once placed an experimental electric tube upon a book, beneath which was a loaded photographic plate holder. The foundation of this story, which the author states to be unknown outside the United States, is traceable to a Chicago scientist who was a student in 1895 at the Wurzburg University and who incorrectly placed the date of the discovery in the spring of 1895. Since Röntgen's two assistants did not know of the discovery till it was publicly announced late in December, it is exceedingly unlikely that any students at the university should have had any insight into the work of their professor. Röntgen's wife, Bertha, was one of the few persons made aware of the discovery for the first time in November, 1895. Another story connected with the discovery is that the first observation of the fluorescent light was made by Röntgen's laboratory servant. This is contradicted by Röntgen's adopted daughter, who now lives in Hanover, and also by a letter of Röntgen himself, dated April, 1921, in which a reference is made to the story, and the inventor asks: "What miserable, envious man has invented this story?" Besides these facts, it is probable that Röntgen's discovery was not by any means accidental, as the great professor, in common with many other scientists—Crookes, Goodspeed, Goldstein, Leonard and others—had for some time been investigating the phenomena of Crookes's tubes. The application of the rays to photography and to medical science followed very rapidly. Röntgen himself apparently gave only one spoken address on the subject, on December 28, 1895. The article reprints some amusing cartoons and verse which appeared in *Punch* early in 1896. A reprint of the first skiagram ever made, that of the hand of Röntgen's wife, is also shown.

Osseous Metastases from Mammary Cancer.

CARLOS HORACIO NISEGGI (*Revista Medica Latimo-Americana*, January, 1931) publishes the results of the radiographic examination of osseous metastases in a case of mammary cancer. Almost all the skeletal bones were involved with the exception of those in the hands and feet. Some of the lesions were hypertrophic in character, but most of them were destructive. There was no increase in size of the diploe of the skull. He makes references to similar metastases from cancers of the prostate, thyreoid, hypernephromata, the œsophagus, uterus, bronchi and stomach, and quotes Adler, who found osseous metastases in 57 out of 374 cases of pulmonary cancer. The radiographic picture may vary so as to simulate either osteomalacia or von Recklinghausen's disease; thus interpretation may be difficult. The author's patient was a woman, aged fifty-eight, who had been operated on in January, 1928, for carcinoma of the left breast with enlarged axillary glands. The last X ray photographs were taken in October, 1930.

Congenital Syphilis.

EDWARD C. VOGT (*American Journal of Roentgenology and Radium Therapy*, July, 1931) discusses the value of Röntgenography in the diagnosis of congenital syphilis. The chief purpose of the study was to ascertain as nearly as possible how far Röntgen findings can be relied on for the recognition of congenital syphilis in infants. The patients reviewed comprised 109 infants under two years of age. The most common complaints were failure to gain weight, or loss of weight, rash or skin lesions, snuffles, paralysis or failure to use one or more of the extremities; less commonly, convulsions, diarrhœa, vomiting or localized tenderness. The Röntgen signs readily recognized and of chief importance are: (i) Bone production as subperiosteal thickening, (ii) bone destruction or osteomyelitis, (iii) disturbance of growth. Subperiosteal thickening was the most frequent finding, but to be of much diagnostic value it should be bilateral and involve many bones. It must be differentiated from periostitis due to other infective processes. Osteomyelitis is usually accompanied by new bone formation. It occurs at or near the end of the long lobes and may involve the epiphyses. A special type consisting of bilateral semilunar defects at the upper ends of the tibiæ, medially adjacent to or just below the epiphyseal lines, is stated to be pathognomonic of syphilis. Disturbance of growth may be demonstrated as a dense, narrow line at the ends of the diaphyses. This is common to all severe nutritional disturbances, but when accompanied by a parallel zone of diminished density, it is very suggestive. Serration of the metaphyseal margins is very characteristic. In the skull irregular areas of destruction or slight thickening of the cranial vault are seen. A common location for skull erosion is along the lambdoidal and posterior portions of the sagittal sutures, with the result that a wide gap occurs in which there are multiple bony spicules.

Gall Bladder Disease.

GEORGE LEVENE (*American Journal of Roentgenology and Radium Therapy*, July, 1931) describes the study of gall bladder contraction as an aid in the X ray diagnosis of gall bladder disease. The author states that there appears to be accumulating experimental and clinical evidence, establishing the fact that the gall bladder contracts and that active contraction is an essential element of gall bladder function. It is impossible to establish an arbitrary

time limit within which the vesicle should completely empty after feeding. In most cases it probably takes place within five or six hours. The time, however, may be as short as one hour or as long as twelve hours, and the emptying time is usually parallel with the gastric and intestinal activity. Functional disturbances affect the emptying time, and the organ is sensitive to reflex stimulation from other digestive organs owing to the sympathetic innervation. Delay in contraction is due to: (i) reflex disturbances accompanied by nausea and vomiting, (ii) improper food stimulation, in which case contractions are stimulated by ingestion of fat, (iii) gall bladder disease, including cholecystitis, stones, and pericholecystitis.

PHYSICAL THERAPY.

The Treatment of Warts.

EARL D. OSBORNE AND EDWIN PUTNAM (*Radiology*, March, 1931), in an article on the treatment of warts, state, and it is common knowledge among dermatologists and other medical practitioners examining large numbers of college students, both male and female, that the last ten years have seen a steady increase in the number of warts, particularly on the feet. This opinion is substantiated by the authors' statistics. Within the past three years 765 patients were examined and treated. The reason for this is the wide popularity of all forms of sports requiring the common use of dressing rooms, shower baths *et cetera* which has increased the probability of infection. The authors' technique for the treatment of plantar warts with X rays is as follows. The patient is placed on a high examining table. The keratotic layer over the wart, and for some distance around it, is carefully pared down with a new razor blade to a point just short of bleeding. Careful attention to this one factor has increased the percentage of cures at least 20%. After all the lesions are located, they are carefully outlined with a fine pointed pen exactly at the periphery of the wart. A sheet of thin oiled silk is placed over the area on the foot and a careful tracing is made of the lesion. This tracing is transferred to lead foil, and the holes are cut out accurately with a sharp, fine scissors. The patient is then placed on his abdomen or side and the foot held in a stirrup which will not move; the stirrup is so fastened as to give as much stability as possible to the entire leg. It is essential that the patient be under no muscular strain while treatment is going on. The lead foil is fitted exactly to the wart area and fastened down by adhesive straps, which go entirely around the foot. After instituting this procedure it was found that no reaction occurred in the normal tissue surrounding the wart with the ordinary dosage of from two to three skin units of X rays. The authors have gradually increased this dosage until they have reached eight skin units with no visible reaction in the normal tissue in a vast majority of cases and only a slight erythema in those that did react. They have not had in the past three years, since following this technique, a single disagreeable reaction from X rays. In treatment with X rays the authors have employed the following factors: A fifteen centimetre (six inch) spark gap, six milliampères of current and a twenty centimetre (eight inch) distance from cathode to skin. The percentage of cures has risen 20% since the single massive dose was employed. The authors have never repeated the dose within two months and have seen warts disappear ten weeks after the one initial maximum dose. They have never given more than two maximum doses to a single wart. Out of a total of 322 patients with plantar warts, 220 were treated with X rays. Of these 176 were cured with one treatment, 80%; 17 patients were cured with two X ray treatments, 7·7%, giving a total percentage of 87·7 cured by X rays alone. Thirteen patients were not cured by X rays and were later operated on with electro-coagulation, all of them being cured. Five patients were not cured by electro-coagulation, but were cured with subsequent applications of X rays. Thirty-nine patients were cured by electro-coagulation alone. Five patients required two operations with electro-coagulation before they were cured. During the past three years only six patients have been treated with radium, with three failures, due to the fact that treatment was unusually difficult. In these three cases cure was later effected by electro-coagulation. Electro-desiccation and electro-coagulation are the methods of choice in the treatment for the majority of patients with warts on the hands, face, neck, genitalia and general body surface. Isolated warts on the hands can be treated with X rays, the same technique being used as in plantar warts, with smaller dosage.

Radium Therapy for Osseous Metastases from Mammary Cancer.

ANGELO SANTORO (*Il Policlinico*, April 13, 1931) records a case of vertebral metastases secondary to mammary cancer which he treated successfully with radium. The patient, aged seventeen, had a cylindrical-celled carcinoma of the left breast which was operated on in April, 1929. In July of the same year she suffered pain in the lumbo-sacral region, and a radiograph revealed a destructive process of the fourth and fifth lumbar vertebræ. In December of the same year severe pain was felt in the right hip joint, and subsequently a spontaneous fracture of the neck of the femur occurred. Deep X ray therapy could not be used because transportation was impossible. An apparatus was made of Columbia paste and moulded over the whole of the pelvis, including the two hip joints. A hundred and fifty milligrammes of radium element in fifteen foyers of ten milligrammes were distributed over the mould. The distance was six centimetres, the filtration two millimetres of platinum, and the dose was delivered over twenty-one consecutive days. After eight days the agonizing pains disappeared. Daily injections were also given of colloidal silicate of magnesium. A subsequent application was necessary. The amelioration in the general and local conditions was remarkable.

X Ray Treatment of Mediastinal Tumours.

F. PORDES (*Wiener Medizinische Wochenschrift*, May 30, 1931) gives his experience with the radiological treatment of various mediastinal growths. First of all, any active tuberculous lesions must be excluded before treatment is commenced. The author emphasizes the importance of commencing with small doses, which should be applied to a small portion of the growth only, until any reactions be noted. The trachea and the area around the bifurcation should be outside the area of radiation because of the risk of sudden death following œdema of the glottis. In extremely radio-sensitive growths there are frequently seen marked general disturbances and fever as well as alterations in the size of the tumour after the first radiation. By observing the above rules such sequelæ are reduced to a minimum. Further treatment as regards dosage and intervals depends on the nature and extent of the growth, and these must be calculated according to the individual case.

Röntgen Therapy in Leuchæmia.

GIULIO CALCHI NOVATI (*L'Ospedale Maggiore*, March, 1931) discusses the value of Röntgen therapy in various forms of leuchæmia in a report of thirteen cases. He considers this the most valuable therapeutic method available, even if it is impossible by its means to cure definitely these conditions. The more chronic the disease, the more efficient is X ray therapy. In acute and subacute cases the treatment is not only ineffective, but may even aggravate the disease. In a favourable case the blood picture improves and the symptoms abate, but after a varying interval relapse occurs and the beneficent effect of the rays grows less with each new application because of acquired radio-resistance. In chronic myeloid and lymphatic leuchæmia, six hours after a *séance* there is a transitory hyperleucocytosis to which succeeds a notable diminution of the leucocytes. X ray therapy often determines a considerable increase in the elimination of uric acid, an index of cellular destruction. The curve of elimination reaches its highest after a certain number of *séances* and then descends gradually to normal. Small and medium doses were given over the whole of the hæmopoietic system—spleen, lymph glands, long bones and liver.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SECTION OF MEDICINE OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the British Medical Association House, Macquarie Street, Sydney, on June 18, 1931, PROFESSOR C. G. LAMBIE, the President, in the chair.

Angina Pectoris and Coronary Occlusion.

DR. C. B. BLACKBURN read a paper entitled: "Angina Pectoris and Coronary Occlusion" (see page 375). The paper was illustrated with electrocardiograms which had been prepared by DR. A. J. HOOD STOBO. Dr. Stobo explained the characteristic features of each tracing. He said that the electrocardiogram might be described as a graphic representation of the variation of electrical production coincident with the contraction and relaxation of the muscles of the different chambers of the heart. In the absence of any electrical change consequent on the contractions of these muscles the line would show no deviation upward or downward, in other words, it would remain in what was called an iso-electric position.

The *P* wave was caused by the electrical production during the contraction of the muscles of the auricles. The *Q R S* group was caused by the alteration of potential during the spreading of the contraction over the ventricles.

The causation of the *T* wave was as yet not thoroughly understood. It was caused by the alteration in potential during the latter part of ventricular systole, and might be due either to the coincident contraction of all the muscle fibres of the ventricles or to the fact that certain of these fibres relaxed before others, or to a combination of these two processes.

The *P* wave was found as a summit in all normal and young adult subjects in Leads I and II, but might be diphasic or inverted in Lead III.

The variations in the *Q R S* complex were many, even in normal hearts; its total duration should not exceed one-tenth of a second.

Under normal conditions the *T* wave was always upright in young and healthy individuals in Leads I and II, but it was often inverted in Lead III.

The interval after the *Q R S* group and before the onset of the rise or fall of the first part of the *T* wave should be one of slight or no deflection. According to Pardee, this deflection, if present, in normal records was never over one millimetre, and usually was not so much as this.

Dr. Stobo showed the following table, adapted from Pardee, and said that it might be taken as a guide as to what constituted a normal electrocardiogram.

In health the form of the electrocardiogram remained remarkably constant in any one individual. After myocardial infarction, however, striking changes occurred.

Apart from the changes, due to the occurrence of abnormal rhythms and block in the auriculo-ventricular bundle or its branches, which were sometimes found, other important changes of the ventricular complex were present in the great majority of cases.

In its extreme form the type of ventricular complex to be described was absolutely pathognomonic of myocardial infarction, though minor degrees of variation from the normal, in which the T wave took off slightly above the base line, had been met with in other conditions.

During the stage of myocardial necrosis, soon after the infarction, a deviation in position of the *R T* or *S T* period might take place. This might be found either above or below the iso-electric level. Leads I and III showed the deviation best as a rule. In these two leads the deviation would be in the opposite direction. If it was upwards in Lead I it would be downwards in Lead III. An *R T* elevation in Lead I would appear as an *S T* depression in Lead III. This very marked change, however, was unstable and in the course of a few days or weeks gave place to curves in which *T* was inverted in one or other lead. One of the electrocardiograms shown illustrated this transition stage remarkably well.

Owing to the fact that it was not always possible or prudent to take an electrocardiogram immediately after infarction, the later changes were the ones which would be found most commonly.

Pardee, who was the first to describe these changes, stated that the characteristic feature was the upward convexity of the *S-T* interval. The *T* wave in one or more leads was turned downward, often sharply peaked, and showed an upward convexity of the *R-T* or *S-T* intervals. Pardee stated that if this feature was found only in Lead III it could not be considered significant unless *T*2 was also turned downward—the upward convexity need not be present in this lead also.

This change might be permanent or after many months the normal form of the electrocardiogram might be restored, as would be seen in one of the records shown.

DR. S. A. SMITH, in opening the discussion, expressed his indebtedness to Dr. Blackburn and Dr. Stobo for their instructive communications. Undoubtedly the recently acquired knowledge of coronary occlusion had supplied more evidence for the study of cardiac pain. He had been interested to observe that Dr. Blackburn had declared himself an adherent to the "coronary" school of thought in regard to the origin of the pain of *angina pectoris*. Recently the majority of people interested in this subject had come to regard all pain of great severity as due to disease of the coronary vessels, but there were still some who adhered to the old school and believed in the aortic origin of cardiac pain. Many had asserted that the recent knowledge of coronary thrombosis added great support to the "coronary" theory, but he was of the opinion that the contrary was the case and that the theory of Clifford Allbutt, modified by Wenckebach and elaborated by Reid, was not yet defunct. There were two main types of severe cardiac pain; one of these had been fully described by Dr. Blackburn and illustrated in his case histories of coronary occlusion, but there was another and less serious one to which the term angina could be applied, though he agreed with Dr. Blackburn that it was time the word angina was discarded in favour of some more suitable term. *Angina pectoris* occurred after exertion, the pain of coronary occlusion occurred characteristically while the patient was at rest; during an attack of *angina pectoris* the patient was rendered immobile, while the person afflicted with a coronary occlusion was intolerably restless; other manifestations of coronary occlusion were shock, pyrexia and leucocytosis; *angina pectoris* had none of these characteristics. But it was the effects on the

Wave.	Largest Height in any Lead.	Direction.			Form.	Duration.
		Lead I.	Lead II.	Lead III.		
P	1–2 mm.	Upward.	Upward.	Upward diphasic or downward.	Rounded or may be notched.	Not more than 0·10 second.
Q R S	7–16 mm.	Chiefly upward (R).	Chiefly upward (R).	*Q S* or both may equal but not exceed *R*.	One, two or three sharply pointed peaks.	Not more than 0·10 second.
T	1–5 mm.	Upward.	Upward.	Upward diphasic or downward.	Peaked.	—

Q R S-T interval varies with heart rate 0·32-0·42 second and is practically iso-electric.

heart and circulation to which he wished to draw special attention. Coronary occlusion resulted in very grave effects on the heart. There were a fall in blood pressure, a weak pulse, inaudibility of the cardiac sounds, and arrhythmia. There was a serious affection of the function of the heart. In *angina pectoris* there was none of these things. If there were any change in the blood pressure during *angina pectoris*, it was a rise rather than a fall, but there was usually no change. There were no known electrocardiographic changes as a result of *angina pectoris*. There was no evidence of any change in the function of the heart. As Clifford Allbutt characteristically put it, "amid the agitation of patient and attendants, the heart, assumed to be the protagonist in the conflict, often seems to be the one impassive actor." The adherents of the "coronary" theory maintained that both of these sharply contrasted types of pain were due to the same condition. Dr. Smith found it hard to accept this hypothesis. In other organs, when intermittent claudication or spasm of an artery occurred, it produced a definite effect on function. If such an event occurred in the artery supplying a limb muscle, pain was felt certainly, but the animal limped. If the blood supply to a portion of the brain were temporarily interfered with, there was a definite disturbance of function. Surely, therefore, a temporary blockage of a coronary artery should cause a disturbance of cardiac function, but during *angina pectoris* there was no evidence of such a disturbance. Pathological studies appeared to have been of little assistance. The pathologist was unable to say after a patient's death whether he had suffered from *angina pectoris*. Those who had supported the "coronary" theory, said that coronary disease could always be found *post mortem* in persons who had suffered from *angina pectoris*, but it had been denied by good authorities that this was always the case. On the other hand, Dr. Smith had yet to hear that anyone had disproved Allbutt's claim that aortic disease could always be found in such instances when examination was carried out thoroughly. Dr. Smith referred to Reid's views and went on to say that since *angina pectoris* usually followed muscular exertion, it was important to bear in mind certain physiological facts. During exercise the blood vessels of the voluntary muscles dilated; this resulted in a reduction of pressure in the aorta. It was easy to suppose that, having regard to the pathological conditions so often associated with *angina pectoris*, this mechanism was out of gear and that a sudden abnormal rise in endaortic pressure resulted, causing stimulation of the sympathetic nerve fibres and pain. Thus it might be said that there were two causes of cardiac disease. Was there any way of distinguishing between the two? Perhaps as knowledge increased it would be possible to do this. In the light of existing knowledge there were some suggestive facts. The pain of *angina pectoris* was felt behind the upper part of the sternum, that associated with coronary disease was felt behind the lower part of the sternum and even in the epigastrium. *Angina pectoris* was often relieved immediately by the administration of nitrites, presumably as the result of a fall in aortic pressure, produced indirectly; the blood pressure, the character of the pulse *et cetera* were also of assistance in differentiation, and electrocardiography might also prove helpful. Dr. Blackburn had shown an admirable series of case histories. Dr. Smith proposed to describe one more, illustrating the fact that coronary occlusion could occur with relatively slight symptoms. A man, seventy years of age, while sitting at his desk suddenly felt ill and nauseated and noticed that his pulse was irregular. He walked into the neighbouring room of a colleague to tell him that he proposed to discontinue work for the day. His colleague remarked that he looked deathly pale. Next day the electrocardiograph revealed clear evidence of coronary occlusion. A transient auricular fibrillation occurred a few days afterwards, but no symptoms had manifested themselves for many months. At the time of the meeting the patient was in as good health as previously, though the electrocardiographic appearances still contained traces of evidence of the coronary occlusion. With regard to electrocardiography, neither Dr. Blackburn nor Dr. Stobo had dealt with the question of localization of the cardiac lesion. Recent observations seemed to justify the hope that it would be possible eventually to localize coronary lesions as accurately as cerebral lesions. Dr. Smith referred briefly to Graib's work on the *T* wave and its application to the interpretation of the electrocardiogram in coronary disease. He wished finally to refer to the methods of some pathologists in examining the coronary arteries; these were often crude and clumsy. Very often the pathologist's report that there was no evidence of coronary occlusion was due to inadequate examination.

DR. A. W. HOLMES À COURT said that the address had been an admirable elucidation of some of the problems of cardiac pain with which medical practitioners were not infrequently confronted. Stress had rightly been laid on the importance of the recognition of intercurrent factors simulating or aggravating cardiac symptoms. It was, for example, a matter of not uncommon clinical experience to find attacks of pain, apparently anginal in character, associated with gall bladder disease in females, the cardiac symptoms being greatly relieved when the offending gall bladder was removed, in that the cardiac pain was then much less likely to be experienced. The explanation of of the "summation" effect, which seemed to render patients suffering from other conditions producing painful sensations referred to the areas of distribution of the cervical and upper dorsal roots unduly prone to pain, apparently of cardiac origin, and even to definite anginal attacks, was somewhat obscure. It would, however, seem not improbable as a tentative hypothesis, that this was really an example of the "phenomenon of facilitation" or, in other words, that the pain path or reflex arc, having been rendered unduly sensitive by painful stimuli, a subliminal stimulus arising from the cardiac mechanism eventually became an adequate stimulus for pain production. It was probable also that the reflex mechanism might be stimulated or sensitized by psychic or toxic factors as well as by reflex causes; of such a nature was tobacco angina. Appreciation of these facts was of great value in assessing prognosis in patients affected with cardiac pain, when it was realized that in such cases a severe painful response might be elicited by a comparatively insignificant cardiac disturbance if the "pain path" had been otherwise unduly excited. In cases of this nature treatment of the associated pathological condition was often effective in bringing about a gratifying diminution in cardiac symptoms, and the outlook was consequently much better than when the symptoms were essentially and entirely cardiac in origin. Those present were indeed under a debt of gratitude to Dr. Blackburn for putting this aspect of the matter so plainly before them.

DR. J. COEN agreed with Dr. S. A. Smith that it was still correct to retain the aortic theory of *angina pectoris*. Not only that; the theory might be extended to the coronary arteries. The association of pain with effort led to the inquiry as to the circulatory changes produced by effort. Effort raised the blood pressure and caused an increase in the minute volume of the blood; such an increase meant an increased coronary blood flow. Starling had shown that with increase in the blood pressure the flow through the coronary vessels might be increased tenfold. At the end of the experiment one-third of the whole of the blood in the body was passing through the heart per minute. A realization of this fact showed what efficient conduits the coronary arteries needed to be. Overdistension, as opposed to spasm, might lead to pain. This theory raised the question as to whether there was arterial pain in the same sense as there was intestinal or ureteral pain. Measures adopted for the relief of angina had a bearing on the question. The administration of amyl nitrite would relieve pain by reducing the blood pressure and relieving the strain on the coronary arteries. Adrenalin, on the other hand, by raising the blood pressure and dilating the coronary arteries would precipitate an anginal attack. This would explain why emotional excitement might cause an attack of angina, because every state of emotion or excitement was accompanied by an increase of adrenalin in the blood. Amyl nitrite did not relieve pain due to coronary thrombosis because there was the added factor of the thrombus which perpetuated the abnormal

state of the coronary artery. The age incidence of coronary thrombosis was of interest. The ages of the patients in Dr. Blackburn's series averaged about fifty-five years. It seemed undoubted that coronary thrombosis occurred at a much earlier age than had been supposed. Electrocardiographic changes were definite in coronary thrombosis. They probably had some relation to the architecture of the coronary vessels. The time factor was also of importance; the typical change might be late in appearing.

DR. A. E. MILLS offered his congratulations to Dr. Blackburn for his paper and the wealth of material which he had gathered and presented to the meeting. He wished also to congratulate Dr. Stobo for his clear exposition of the electrocardiogram shown. With reference to the subject of the paper, his remarks would be confined to the causation and source and distribution of pain in *angina pectoris* and coronary occlusion. There was abundant evidence to show that deficient blood supply to the skeletal muscles resulted in pain when such muscles were working. When the muscles were at rest there was no pain. Krogh had shown that when a muscle was at rest, a great number of the capillaries going to its fibres were in a state of complete contraction, preventing any blood passing through them. On the other hand, when the muscle was working all these closed-down vessels opened up and a great and sufficient blood supply was then available. It had been shown by Krogh that in the muscle of the guinea-pig about twenty times as many capillaries were opened during activity of the muscle as during rest. Such an enormous increase in the volume of blood going to muscles when working provided for the greatly increased oxygen requirements of the working muscle. With the muscle at rest oxygen consumption was very low and only a limited blood supply was needed. It was essential to bear in mind this alteration and adjustment of oxygen requirements to muscles at rest and at work. If from any cause the adjustment was at fault and an imperfect supply of oxygen was thereby not available for the working muscle, pain resulted, and such pain might surely be considered as due to oxygen want. Such a failure occurred in that condition known as intermittent claudication, where pain was experienced in the muscles of a limb when they were working, but was absent when the muscles were at rest. Further, the pain increased as the exercise continued and forced the patient to rest.

The speaker then cited characteristic cases of this disease which he had seen, and said that it seemed reasonable to believe that many cases of anginal pain of cardiac origin were due to this same cause, namely, the failure to adjust the oxygen supply to the varied requirements of the cardiac muscle.

Whether the closing and the opening of capillaries leading to a less or greater oxygen supply, according as the muscle was at rest or at work, was dependent upon a nervous reflex arc, or due to metabolic chemical stimuli was not yet clear, but the evidence pointed to the latter as a more probable cause. In any case the evidence that the pain of *angina pectoris* was in the majority of cases due to vascular disturbance was almost overwhelming.

With regard to the site and distribution of the pain in *angina pectoris* and coronary occlusion it must have the character of so-called visceral pain; in other words, it must be felt in those segmental areas of the body in relation to the spinal segments with which the viscus was in nervous connexion. As the heart was connected with the upper three or four thoracic segments, impulses arising in it must first pass to those segments. If these impulses were sufficiently severe as to give rise to pain, then the pain would first be felt in the corresponding somatic segmental areas.

The pain impulses might be very severe. They might then spread by means of intersegmental neurones to adjacent spinal segments and pain would then be felt in the corresponding somatic segmental areas. So strong painful impulses originating in the heart might overflow to all the cervical spinal segments and most of the thoracic spinal segments. In such a case the patient might complain of pain in his lower jaw, all over his neck and chest and arms and over the abdomen. But, and this was of fundamental importance, in the first place it would be felt in the region of the chest. On the other hand, strong impulses from the abdominal viscera, after reaching those spinal segments with which they were in connexion, might overflow to higher and lower spinal segments. The pain first experienced over lower thoracic segmental areas might spread and be felt over those areas which were so commonly the seat of pain in cases of *angina pectoris*. But in such cases Dr. Mills did not think there was sufficient justification for saying that the patient was suffering from *angina pectoris* or that the abdominal visceral disorder also gave rise to *angina pectoris*, because the pain of *angina pectoris* arose from the heart and was due to some disease or disorder of that organ.

It was then highly important to know the spinal segments which were in nervous connexion with the various organs and their corresponding somatic segmental areas. By means of such knowledge it was possible to say where pain arising from any organ would first be felt and what organ might be affected when pain was felt over certain definite somatic segmental areas.

DR. M. C. LIDWILL added his congratulations. If interference with the coronary supply was the cause of pain, why was it that, in certain cases of coronary occlusion, there was no pain whatsoever. He had seen three cases in which there had been no pain at all. In one instance a man, aged thirty-two years, while working in the garden, was seized with dyspnœa and brought to hospital, where he remained for a period of six weeks. He was allowed to go out, and returned afterwards and died of cardiac failure. His condition was diagnosed prior to death by means of electrocardiography, and the diagnosis was verified by *post mortem* examination. If anoxæmia caused pain in some instances, why not in every instance? The coronary circulation changed with increasing years, and as a man became older the anastomoses of his coronary vessels became more profuse, so that the older the person afflicted with coronary occlusion, the greater the likelihood that he would recover. Dr. Lidwill referred to the foramina of Lannalongue and the Thebesian system of blood ducts in the heart, explaining that these communicated with the coronary veins and were filled during diastole and emptied during systole. He suggested that this auxiliary circulation might have some effect in preventing anoxæmia, and hence pain. He mentioned several illustrative cases.

PROFESSOR C. G. LAMBIE remarked that those present could congratulate themselves on the privilege they had enjoyed of listening to such able communications and such an illuminating discussion. The recognition of the syndrome of coronary occlusion was one of the most important advances in cardiology within recent years. The correlation between the work of the clinicians, the morbid anatomists and the experts in electrocardiography had been along the correct lines and had resulted in an advance of knowledge of definite value in diagnosis and in prognosis. But there still remained many unsolved problems. Very little was known with regard to the ætiology, the nature of infections of blood vessels, or the toxins which caused changes in the vessels. Dr. Blackburn had drawn attention to the association of spondylitis and gall bladder disease with cardiac pain. No doubt the believers in focal sepsis would see in such cases evidence of the truth of their theories and would regard the cardiac pain as due to changes in the heart arising from focal infection. Little was known of the nerves concerned in cardiac pain or of the reflexes concerned in the control of the coronary circulation. The controversy concerning whether the pain arose from the aorta or the myocardium was not finally settled. John Wiley had remarked that *angina pectoris* was the "cry of the heart for more blood," and the theory of relative ischæmia of the heart wall was still in favour. Adrenalin was an important physiological reagent in these investigations, for it caused dilatation of the coronary arteries but constriction of arteries elsewhere.

Dr. Blackburn, in reply, expressed his thanks for the appreciative remarks that had been made by various speakers. Dr. Smith, in questioning the coronary origin of *angina pectoris*, had contrasted the usual appearance of these attacks after exertion with the frequent occurrence of occlusion during rest. But surely in an almost

obliterated vessel the final plug of clot would be most likely to form when the circulation was at its lowest ebb. In regard to the absence of pyrexia and leucocytosis in *angina pectoris*, these could hardly be expected unless the lesion producing the symptom complex was an inflammatory one. He agreed with most of Dr. Mills's remarks, but thought Dr. Mills had misunderstood what he had said about the association between *angina pectoris* and disease of the gall bladder. He did not believe that the gall bladder would initiate angina in a normal heart, but that it could start an attack when through cardiac disease the spinal cells receptive of visceromotor stimuli were abnormally sensitive. In reply to Dr. Coen he remarked that he had indicated in his paper that the fact that adrenalin caused angina in the presence of cardio-vascular disease suggested a breakdown in the depressor mechanism through which in health the coronary circulation was protected from the vaso-constrictive action of the drug.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Kee, Harry, M.B., B.S., 1928 (Univ. Sydney), 64, Arden Street, Clovelly.
Saunders, Arthur Clive, M.B., B.S., 1931 (Univ. Sydney), Parramatta District Hospital.

Correspondence.

AN APPEAL.

SIR: In connexion with the effort which I am making to carry on and keep up to date the valuable collection of Australian medical literature presented to this library by Dr. J. H. L. Cumpston, I received from the Adelaide Hospital seven issues of *The Medical and Scientific Archives* of that institution. Unfortunately the hospital was not able to supply numbers 1 (1922) and 7 (1928), as these are out of print.

I should be greatly obliged, therefore, if any reader of your journal who may have these copies available for donation, would get in touch with me, as I am most anxious to complete the series, especially as the hospital has kindly undertaken to see that all future issues are regularly supplied.

Yours, etc.,

KENNETH BINNS,
Librarian.

The Parliament of the Commonwealth,
Canberra,
September 9, 1931.

BUSINESS PSYCHOLOGY.

SIR: Business psychology is apparently being well taught these days.

In common with several of my friends in the suburbs, I have had a visit from a young gentleman who sells ties said to be made from wood fibre, of which process, he says, he has a monopoly; and although he makes them a million or so at a time, he will sell you just one at a cut rate because of the gratitude he owes you for making a "good job" of his poisoned wrist.

He works on the fact that none of us likes to say he does not recognize a patient, and so he claims acquaintance on the score of an illness of his mother, or of a scar on the wrist, which he suggests has been made by the doctor he is interviewing.

I have no idea of the value of the ties, but I feel that if those of us who have not already been "prospected," are forewarned, we may soon persuade him that our little professional peculiarities are not to be used for the base ends of trade.

I feel that he richly deserves Homer's description of Ulysses—*πολύμητις*—for truly he appears to be a man of many wiles, and varies his attack with circumstances. When I blandly disclaimed any knowledge of his wrist he dangled in front of me the bait of a full examination next Thursday, for which he suggested that he was prepared to pay fees on the higher scale. This he said he needed in order to get a certificate of good health, in order to take up an appointment with a well-known shipping company. This he has not said to any of the men I have discussed him with, so that he apparently alters his plans to suit the situation.

I tremble to think what will happen if he turns his attention to my brethren in Macquarie Street, who, undeservedly I am sure, are generally held to be not altogether immune from salesmen's wiles.

On the whole, I think we are to be congratulated that he has confined his attention to selling ties and has not applied his undoubted talents to the netting of higher game.

Yours, etc.,

T. MAYNARD FURBER.

143, Macquarie Street,
Sydney,
September 15, 1931.

CUSTOMS CHARGES ON BOOKS.

SIR: In your leading article of today you venture into the field of economics. As a student of that subject, I feel that certain of your statements ought not to be allowed to pass without comment. You say: "When many books are bought, much money leaves the country." This is certainly not true. International trade is an exchange of commodities for commodities. Not a single book of any sort or description can enter this country unless goods of equal value, made in Australia, are sent out in exchange for it. The student or practitioner who buys a book published abroad, is in the last analysis paying for wool, wheat or butter produced by his own countrymen.

A duty designed to prohibit or restrain the importing of books, boots and shoes, or cheap magazines will have a like effect on the export of such commodities as would have gone away in exchange for them. When one looks at our present fiscal policy and our present plight, is it any wonder that one of our poets, who no doubt would have been "encouraged" by a duty on magazines, was moved to pen these lines:

Wheat in the silos running over,
Food for the worms and the birds that hover,
Food for the rats, for the mice a bed,
And little children crying for bread!

In conclusion let me draw your attention to the duties on drugs and scientific apparatus, which certainly are "prejudicial to the pursuit of scientific knowledge and the treatment of sick people."

Yours, etc.,

KINGSLEY HUDSON.

Bungendore,
New South Wales,
September 12, 1931.

Books Received.

PRIMARY SYPHILIS IN THE FEMALE, by T. A. Davies, M.D.; 1931. London: Humphrey Milford; Sydney: Angus and Robertson. Demy 8vo., pp. 119, with illustrations.

RESTORATION EXERCISES FOR WOMEN, by E. A. Hornibrook, with Foreword by Sir Arthur Keith; 1931. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 106. Price: 5s. net.

THE RATIONAL TREATMENT OF VARICOSE VEINS AND VARICOCELE WITH NOTES ON THE APPLICATION OF THE OBLITERATIVE METHOD OF TREATMENT TO OTHER CONDITIONS, by W. T. Warwick, M.A., M.B., F.R.C.S.; 1931. London: Faber and Faber. Crown 8vo., pp. 187. Price: 5s. net.

CLINICAL OBSERVATIONS ON THE SURGICAL PATHOLOGY OF BONE, by D. M. Grieg, M.B., C.M., F.R.C.S., F.R.S.E.; 1931. Edinburgh: Oliver and Boyd. Demy 4to., pp. 259, with 224 illustrations. Price: 30s. net.

A SYSTEM OF BACTERIOLOGY IN RELATION TO MEDICINE (Privy Council Medical Research Council); Volume VIII; 1931. London: His Majesty's Stationery Office. Crown 4to., pp. 420.

TRAUMATOTHERAPY, THE TREATMENT OF THE INJURED, by J. J. Moorhead, B.Sc., M.D., F.A.C.S. (D.S.M.); 1931. Philadelphia: W. B. Saunders Company; Melbourne and Christchurch: James Little. Royal 8vo., pp. 574, with 625 illustrations. Price: 42s. net.

THE ANATOMY OF THE NERVOUS SYSTEM FROM THE STANDPOINT OF DEVELOPMENT AND FUNCTION, by S. W. Ranson, M.D., Ph.D.; Fourth Edition, 1931. Philadelphia: W. B. Saunders Company; Melbourne and Christchurch: James Little. Crown 4to., pp. 478, with 341 illustrations, some in colour. Price: 39s. net.

CLINICAL DIAGNOSIS BY LABORATORY METHODS: A WORKING MANUAL OF CLINICAL PATHOLOGY, by J. C. Todd, Ph.B., M.D., and A. H. Sanford, A.M., M.D.; Seventh Edition; 1931. Philadelphia: W. B. Saunders Company; Melbourne and Christchurch: James Little. Royal 8vo., pp. 765, with 347 illustrations, 29 in colours. Price: 36s. net.

PRACTICAL RADIATION THERAPY, by I. I. Kaplan, B.S., M.D., with a special chapter on Applied X Ray Physics, by C. B. Braestrup, B.Sc., P.E.; 1931. Philadelphia: W. B. Saunders Company; Melbourne and Christchurch: James Little. Royal 8vo., pp. 354, with illustrations. Price: 36s. net.

NOTES ON RADIUM THERAPY FOR MEDICAL STUDENTS, by H. A. Colwell, M.B., Ph.D., M.R.C.P., D.P.H.; 1931. London: H. K. Lewis. Crown 8vo., pp. 175, with 15 illustrations. Price: 6s. net.

HUMAN HEREDITY, by E. Baur, E. Fischer and F. Lenz, translated by E. and C. Paul; 1931. London: George Allan and Unwin. Royal 8vo., pp. 734, with 172 illustrations in the text and 9 plates. Price: 30s. net.

STUDIES IN THE PHOTO-ACTIVITY AND THERAPY OF THE TUNGSTEN-TITANIUM ARC, by J. Burdon-Cooper, M.D., B.Sc., G.T.V.D.R., D.O., F.C.S., and A. Roberts, T.D., F.R.C.S.E., M.R.C.S.; 1931. Bristol: John Wright and Sons. Royal 8vo., pp. 85, with illustrations. Price: 10s. net.

HAY-FEVER, HAY-ASTHMA, ITS CAUSES, DIAGNOSIS, AND TREATMENT, by W. Lloyd, F.R.C.S.; Third Edition; 1931. London: Straker Brothers. Demy 8vo., pp. 130, with illustrations.

GASTRIC ACIDITY, AN HISTORICAL AND EXPERIMENTAL STUDY, by J. D. Robertson, M.D.; 1931. London: John Murray (for the Middlesex Hospital Press). Demy 8vo., pp. 82.

THE MODERN THERAPEUTICS OF INTERNAL DISEASES, AN INTRODUCTION TO MEDICAL PRACTICE, by A. P. Cawadias, O.B.E., M.D., M.R.C.P.; 1931. London: Baillière, Tindall and Cox. Demy 8vo., pp. 160, with illustrations. Price: 10s. 6d. net.

BRAIN, MIND, AND THE EXTERNAL SIGNS OF INTELLIGENCE, by B. Hollander, M.D.: 1931. London: George Allen and Unwin. Demy 8vo., pp. 288, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

OCT. 1.—South Australian Branch, B.M.A.: Council.
OCT. 2.—Queensland Branch, B.M.A.: Branch.
OCT. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
OCT. 6.—New South Wales Branch, B.M.A.: Council (Quarterly).
OCT. 7.—Victorian Branch, B.M.A.: Branch.
OCT. 8.—New South Wales Branch, B.M.A.: Clinical Meeting.
OCT. 8.—Victorian Branch, B.M.A.: Council.
OCT. 13.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 20.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 23.—Queensland Branch, B.M.A.: Council.
OCT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 28.—Victorian Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Resident Medical Officer (male).

RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Honorary Relieving Assistant Surgeon.

ROYAL NORTH SHORE HOSPITAL, SYDNEY, NEW SOUTH WALES: Medical Vacancies.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

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NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
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SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
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NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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